CLINICO-ETIOLOGICAL PROFILE OF ATRIAL FIBRILLATION IN A TERTIARY CARE HOSPITAL AND ITS CORRELATION TO ATRIAL SIZE

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CERTIFICATE FROM THE DEAN

This is to certify that the dissertation entitled "**CLINICO-ETIOLOGICAL PROFILE OF ATRIAL FIBRILLATION IN A TERTIARY CARE HOSPITAL AND ITS CORRELATION TO ATRIAL SIZE**" is the bonafide work of **Dr. S.SUHAS RAJ** in partial fulfilment of the University regulations of The Tamilnadu Dr. M.G.R Medical University, Chennai, for the award of degree of Doctor Of Medicine (M.D) Branch- I -General Medicine.

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DECLARATION

I, **Dr. S.SUHAS RAJ**, hereby declare that, I carried out this work entitled "**CLINICO-ETIOLOGICAL PROFILE OF ATRIAL FIBRILLATION IN A TERTIARY CARE HOSPITAL AND ITS CORRELATION TO ATRIAL SIZE**" at Kanyakumari Government Medical College Hospital, Asaripallam, under the guidance of **Prof.Dr. PRINCE PIUS M.D.,** Professor of Medicine, during the period of December 2017 to August 2018.

I also declare that this bonafide work has not been submitted in part or full by me or any others for any award, degree or diploma to any other University or Board either in India or abroad.

This is submitted to The Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfilment of the University rules and regulations for the award of degree of Doctor of Medicine (M.D) Branch- I-General Medicine.

Place: Asaripallam

Date:

Dr.S.SUHAS RAJ

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CONTENTS

ABBREVATION

AF **:**Atrial fibrillation

CAD :Coronary heart disease

HF :Heart failure

IVSd :Interventricular septal thickness in diastole

LVIDd :Left ventricular internal diameter(cm) in diastole

LVIDs : Left ventricular internal diameter(cm) in systole

LVPWDd : Left ventricular posterior Wall diameter(cm) in diastole

OSA :Obstructive sleep apnoea

PE :Pericardial effusion

PHT -Pulmonary hypertension

RHD :Rheumatic heart disease

TRPG :Tricuspid regurgitation peak gradient

TTE :Transthoracic echocardiogram

TEE :Transesophageal echocardiogram

INTRODUCTION

Atrial Fibrillation is one of the frequently encountered arrhythmia (weak or irregular heart beat) in our population and it is the deranged supraventricular (atria) event mainly manifesting with irregular heart rhythm which in turn causes altered atrial - electrical and mechanical function. This also has a significant effect on economic burden to the society by causing both morbidity and mortality. Its prevalence though less than 1% in general population below 65 years old ,its incidence and prevalence is in increasing trend of late[1].

Male sex is an important non modifiable risk factor when compared to female sex and additionally its incidence and prevalence are more in males than females. Incidentally females develop atrial fibrillation later in life after sixth decade when compared to male sex who develop bit earlier. White people are more affected than Black People.

A large group of patients affected by atrial fibrillation are initially asymptomatic but in due course they end up with lot of complications, limiting the day today activities of affected population. Due to the abnormality in atrial activity there is abnormal atrial systolic event which leads to a deranged ventricular function with decreased output, leading up to formation of thrombus in atrium which finally ends up in cerebro vascular accident and thrombo-embolic events. Pathophysiology of atrial fibrillation still remains in controversy, but there are many theories that have been proposed like “mother rotor theory” and “multiple wavelet theory”.

There are many comorbid conditions which contribute to the development of atrial fibrillation. Important diseases among them are Rheumatic valvular heart disease, Ischemic heart disease and Systemic hypertension. Smoking habit and alcohol consumption are risk factors adding to the development of such dysrhythmias.

There are different types of atrial fibrillation where causes can be defined, but in undetermined or Lone AF no cause can be found. Different diseases causative to atrial fibrillation will appear at different ages, particularly atrial fibrillation appearing due to valvular heart disease appears earlier than other diseases which contributes to the development of atrial fibrillation.

ECG shows irregular rhythm with normal or rapid rate, absent P waves, normal QRS Complex. Prime treatment method is pharmacological drug treatment while non-pharmacological treatment options are reserved for some patients. Of late newer drugs and approaches under study.

Many researches have been done to find out causes for atrial fibrillation and to frame guidelines for clinicians to decide treatment strategies. Echocardiography is beneficial in finding out the cause for development of atrial fibrillation and various echocardiographic parameters helps in predicting the risk for future development of atrial fibrillation and complications associated with atrial fibrillation. This study is intended to find out the varied presenting symptoms of AF and also possible underlying predisposing factors-cardiac and non-cardiac in Indian context.

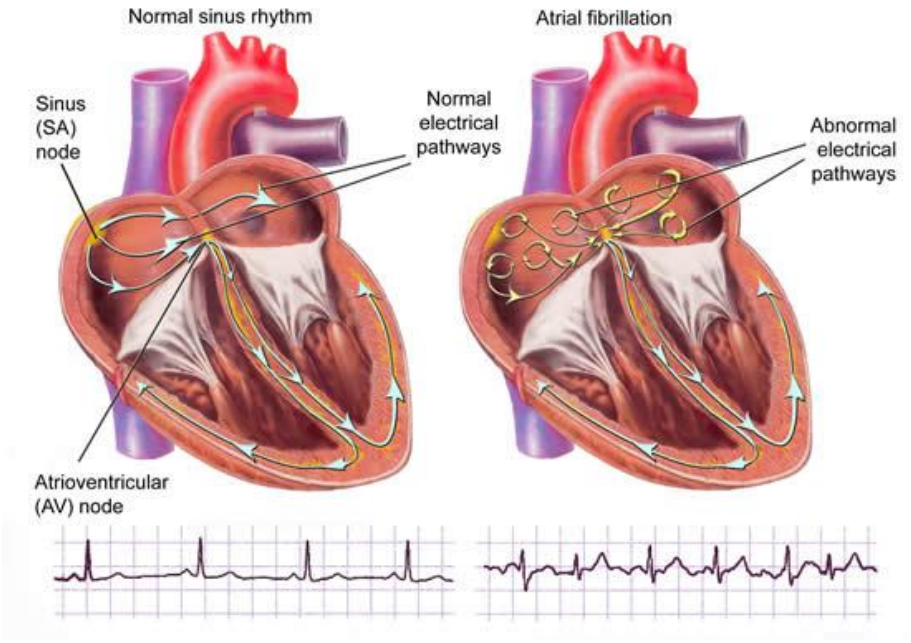
AIMS AND OBJECTIVES

- To study the various clinical presentations of atrial fibrillations admitted in Kanyakumari Govt. Medical College Hospital
- To assess the frequency of underlying heart disease in patients with atrial fibrillation
- Correlation with atrial chamber size with the help of echocardiography

REVIEW OF LITERATURE

Atrial fibrillation is basically a Supraventricular tachyarrythmia with uncoordinated activity of atria characteristically resulting in disordered atrial mechanical and electrical function[1].Electrocardiographically characterized by low amplitude baseline oscillations called as Fibrillatory or 'F' waves and an irregularly irregular rhythm of ventricles with the rate of 300 to 600 beats per minute and they are variable in amplitude, shape, and timing.

Picture 1:



Atrial fibrillation has been involved with the careers of many great clinicians and researchers of twentieth century[2].The first description of atrial fibrillation was mentioned in "Harmony and Health in the Huang TiNei Ching Su Wen"(The Yellow Emperor's Classic of Internal Medicine).He is the physician as well as emperor[3].The term "Fibrillation" was first coined after demonstration of fibrillation of auricles by William Harvey in animals. Jean Baptist de Senac later described relationship between palpitation and stenosis of mitral valve. In 1827 in "Dublin Master of Clinical Expression" Robert Adams mentioned about the association of irregular pulse and mitral stenosis. The first person who put out human ECG portraying atrial fibrillation was William Einthoven in the year 1906.Sir Thomas Lewis who was known as "Father of modern ECG" described about elctrophysiological characteristics of AF whose basic mechanism is circus movement of electrical impulse that is "Re-entry". In the 20th century various clinical features and pathophysiology was described by karel Fredrick wenkebach, Gordonmoe, Bernhordlown and maurits Allessie[4].

EPIDEMIOLOGY:

Atrial fibrillation is the most common type of arrhythmia encountered and treated in current practice. Among arrhythmia related admissions in hospital 33% are due to AF. Atrial fibrillation should be given its due importance due its worldwide distribution which is on the rise and it is the commonest arrhythmia. Indeed renowned scientist Braun wald once in his studies insisted on "growing Epidemic" of AF[5].There is increase in CVA by 5 fold and cardiac failure[6] by three fold, which in turn increases morbidity and mortality. In our country there

is no proper epidemiological data available on AF, but in recent times there is a data about Indian patients from RELY and REALIZE Studies (Indian patient cohort). IHRS-AF registry is the largest study in Indian population in relation to atrial fibrillation[7].

Incidence of Atrial fibrillation is related to age and sex and the distribution is such a way that while the prevalence is around 0.1% per year before the age of 40 years, it is higher than 1.5% per year in female population and higher than 2% per year in males older than 80 years. In the western country prevalence of AF is 1.5-2% in general population, and the average age of presentation of disease is 75-85 years, When annual incidence was analysed it is 3.1 in males and 1.9 in females per 100 person years in persons whose age are below 65 years [8] .From one another population based research done recently in patients older than 65 years the prevalence in women is 4.85% lesser than that of men 9.1%[9]. In Framingham heart study the results were in such a way that AF developed 1.5 times more in males than in females.

Similarly when obesity is associated with increasing age there is a 60% increase in AF Incidence. Due to absence of prominent symptoms incidence and prevalence of AF is often underestimated and its one of important limitations in understanding the epidemiology of AF.

The first study regarding AF to be done in India was way back in 1995 which included around 984 patients. The study showed a prevalence of 0.1%. The study had a lower prevalence because those who were recruited for study were healthy and was subjected to only single ECG, and around 6% of them

were above 65 years old. Another study famously known by name west Birmingham study showed a prevalence of 6% among Indians. Another community based study showed age adjusted incidence of AF per 1000 person years increased between 1980 and 2000 from 4.4 to 5.4 in men and from 2.4 to 2.8 in women [10].

AF is also mainly associated with double the time risk for all cause of mortality. Mortality increases in a person with AF even if he is normal otherwise. "Framingham study" shows Atrial fibrillation independently associated with increase in risk of mortality in men (OR: 1.5; with 95% CI: 1.2-1.8) and women (OR: 1.9; 95% CI: 1.5 – 2.2).

Similarly AF is also linked with fivefold increase in risk for Stroke[11], A study done revealed that globally AF may be the basic reason behind around 75000 strokes per year and also it is one of the important causes of embolic stroke. When associated with other co morbidities like diabetes, hypertension and CAD there is further increase in chance of development of Stroke.

PATHOPHYSIOLOGY:

Anatomical modifications ending up alteration of atrial architecture possibly leads to progression of AF[12.]These structural changes include mainly inflammation, hypertrophy and fibrosis. These changes occur particularly due to basic cardiac disease like as follows

- Coronary Heart Disease
- Valvular Heart Disease
- Hypertension

- Heart Failure And
- Cardiomyopathies

All above said conditions causes dilatation of atrium, changes or increase in wall stress, raised left atrial pressure. Similarly ischemia of atrium due to underlying coronary artery disease and other conditions like

- Hemochromatosis
- Amyloidosis
- Sarcoidosis

Also have high chances of developing AF. The finding supporting this was done in a study where patients who had paroxysmal AF without any underlying structural heart disorder was evaluated with biopsy from atrium which revealed infiltrate of inflammation consistent with fibrosis and myocarditis[13].

Other factors which have pathologic effects on cellular function and atrium are extracardiac conditions like

- Systemic hypertension
- Obesity
- Obstructive sleep apnoea syndrome
- Hyperthyroidism
- Alcohol and
- Drugs.

Most common and repeated pathologic feature seen in AF is

1. Atrial fibrosis
2. Atrial muscle mass loss
3. Inter nodal tract muscle loss.

Mild to moderate fibrosis occurs in patients with AF of lesser duration whereas loss of muscle mass and severe fibrosis occurs in chronic AF. Myocardial fibrosis is the predominant feature of atrial fibrillation commonly seen in both human and experimental animal, atrium is very sensitive to pro-fibrotic signalling pathways than that of ventricles because it contains large quantity of fibroblasts and hence these changes happens predominantly in atrium.

Rapid ventricular pacing in experimental animals yields atrial fibrosis thereby making them susceptible to AF, similarly during rapid atrial pacing also fibrosis occurs. Rapid pacing of atrium for extended periods increases susceptibility to AF. These rapid pacing causes alteration in mitochondria, loss of myocyte from glycogen deposits and abnormal gap junction which lead to Apoptosis and cell necrosis[14].For identification and quantification of the fibrosis[15]best used investigative method is non-invasive gadolinium enhancement MRI. This fibrosis has strong correlation with the development of stroke which is proven by many studies [16].

Atrial dilatation also occurs due to systemic hypertension, coronary heart disease, dilated cardiomyopathy while all these factors leads to stretching of myocardium, which in turn activates various molecular mechanisms like Renin-

Angiotensin-Aldosterone pathway, Angiotensin-II, TGF-α. These mechanisms in turn induce inflammation and production of connective tissue growth factors and fibrosis. Microscopic and macroscopic changes appear in the atrium as early as first year of life. During 4^{th} to 5^{th} decades droplets of fats appears in the region of Atrio ventricular node and septum, whereas in much older persons ageing results in myocardial fibres loss and fatty deposition. Histologically Patchy fibrosis with juxtaposed normal atrium contributes to such heterogeneity of atrial conduction.

In rheumatic heart disease patient we can see the presence of Ascoff bodies pathognomonic of RHD, Anitschkow cells otherwise called as Caterpillar cells, Fish mouth appearance of valves are present particularly involving atria.

MECHANISMS OF ATRIAL FIBRILLATION:

There are lot of mechanisms which adds up to the development of AF, this mechanism may differ in each patient while sometimes all mechanisms may coexist in an individual patient. Mechanisms causing the development of AF and maintaining its course is more complex. The events which trigger this may differ from maintenance mechanisms. AF is more commonly initiated by small re-entrant or rapidly firing focus in the sleeves of atrial musculature along the pulmonary veins.

The clinical Phenotypic variation of atrial fibrillation are

- Paroxysmal
- Persistent and
- Chronic AF

have electro physiologically different features, because of remodelling and clinical modulators of different variety that affect the factors such as atrial stretch, myocardial ischemia, cardiac failure, inflammation, fibrosis, sympathetic and parasympathetic influences. There are basically two principle electrophysiological mechanisms which are the reasons behind development of such fibrillatory activity.

They are Automatic, triggered or foci of micro re-entry called as Drivers. They fire at rapid rates and produce fibrillatory activity. Multiple Re-entry circuits that meanders the entire atrium annihilates and reforms wavelets which ultimately leads to accentuation of fibrillatory activity.

In many instances these two mechanisms may be present together. Studies show that the site of dominant frequency of discharge is in left atrium, with a left to right gradient. One of a study done recently in which patients were subjected to multiple electrocardiograms recorded simultaneously associated with signal processing technique by obtaining computerized maps revealed foci of fibrillatory sources and electrical rotors. In that study a mean of 2.1 sources was found in 97 % of 101 patients, of which 30% was Focal sources and 70% was Motor Rotors[17].

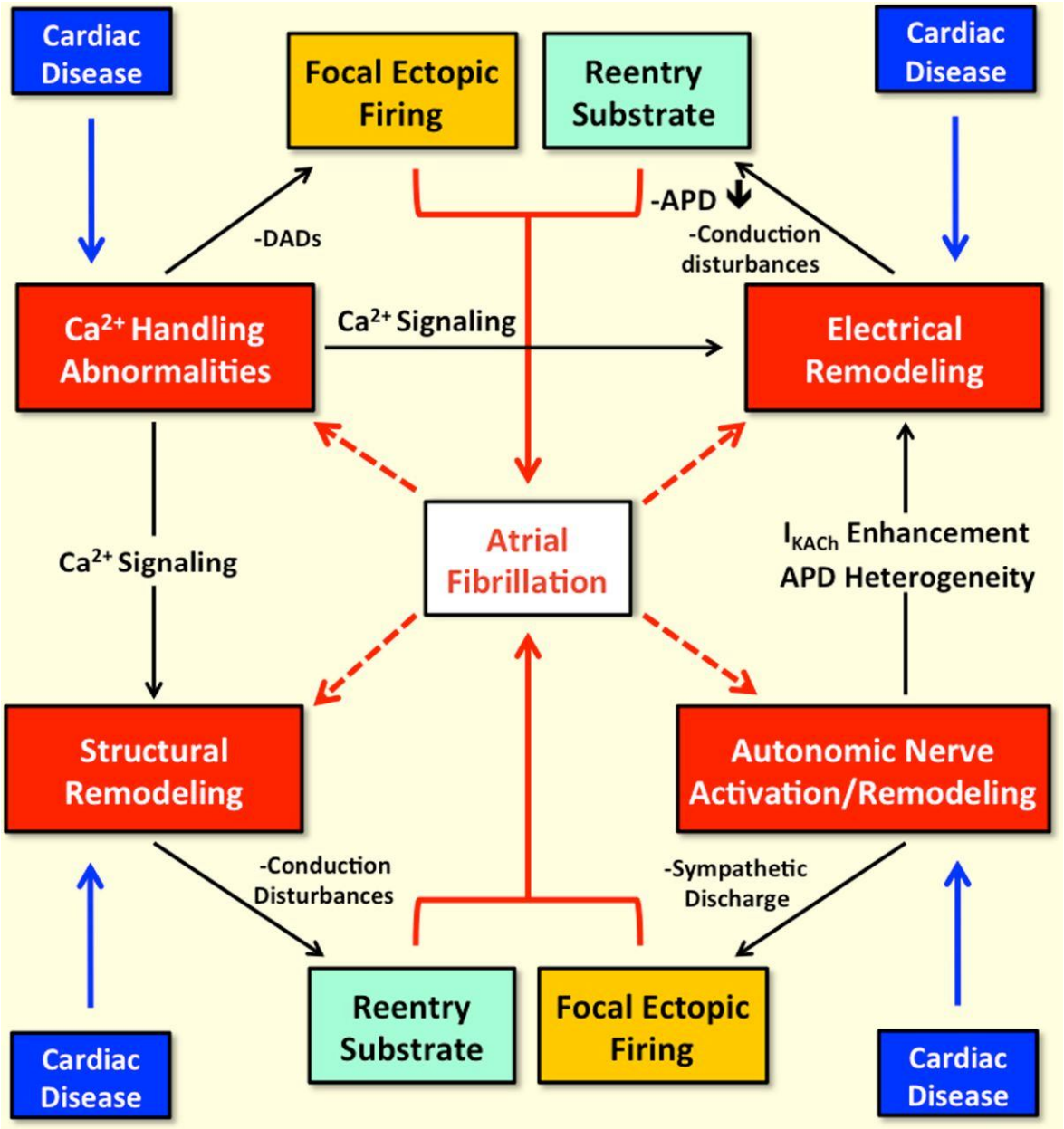


Figure1: Principal atrial fibrillation (AF)–maintaining mechanisms.

MECHANISM OF TRIGGERS IN AF:

Ectopic Focal discharges are often the provoking event in the development AF[18]. Anatomical and electrophysiological specific characteristics of atriopulmonary vein junctions and pulmonary veins are mainly guilty for arrythmogenic tendency. Particularly in patients with paroxysmal AF rapid foci of discharges often arises from sleeves of myocardium of LA that extend to

pulmonary veins. Hence this mechanism makes the basis for the radio frequency catheter ablation by doing pulmonary vein isolation.

Re-entry mainly occurs because of conduction disturbances which leadto comparatively depolarized resting potentials in pulmonary vein myocytes, refractoriness in pulmonary veins and shortened action potentials. All these three changes favour the development of re-entry[19]. Sometimes pulmonary vein myocytes causes abnormal automaticity and trigger alone.

Other sites for development of ectopic foci of discharges includes

- Coronary sinus
- Septum
- Venae cavae
- Posterior LA
- Marshall Ligament.

There are other sources of abnormal activity triggers present in pulmonary veins, which includes

1.Interstitial cells[20],

2. Melanocytes[21],

Other conditions which cause a delayed after-depolarization include intracellular calcium metabolism derangement due to diastolic calcium leak from sarcoplasmic reticulum. This also plays an important role in the occurrence of AF[22].

COMPLEX FRACTIONATED ATRIAL ELECTROGRAMS:

In persistent type of AF atrial substrate changes including interstitial fibrosis which give rise to slow, anisotropic and discontinuous conductions lead on to "complex fractionated atrial electrograms" and re-entry.

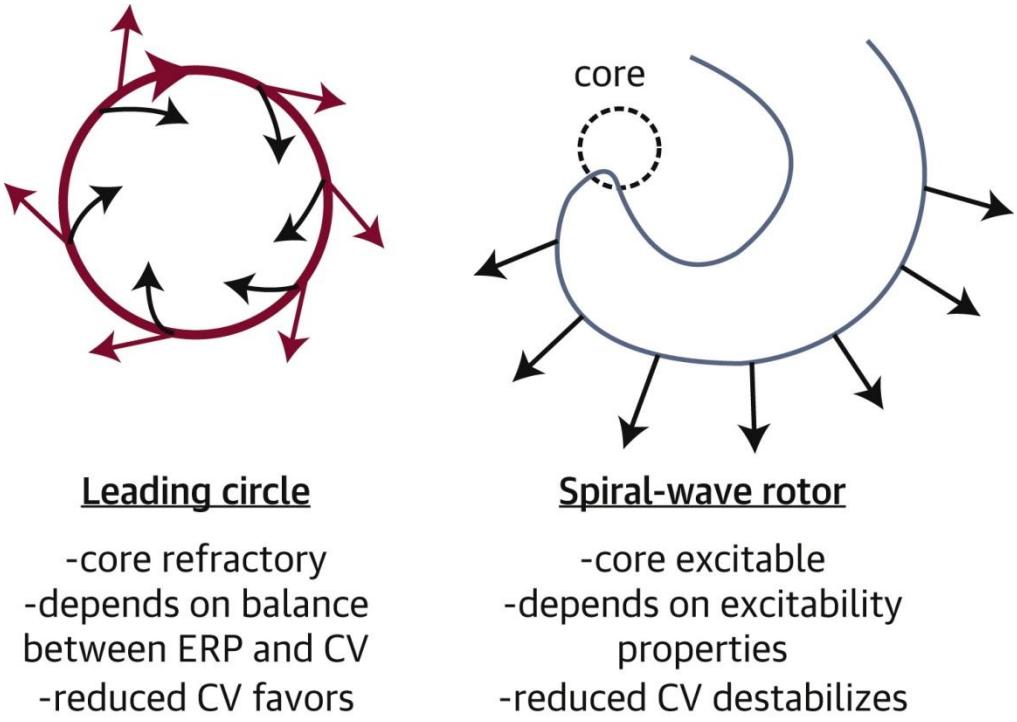


Figure 2: Models of re-entry. A. Leading circle, B. Spiral wave re-entry

FACTORS RESPONSIBLE FOR MAINTENANCE OF AF:

The following theories give explanation to the maintenance AF which includes

1.Rapidly firing foci, which is responsible for activity from ganglion plexus present in the cardiac tissue.

2.Re-entrant wavelets of independent in nature, in multiple quantities associated with conduction and refractoriness of heterogeneous nature.

3.Spiral wave Re-entrant circuits or, Rotors[23].

Rotor excitation with a single rapid focus, leads to refractory tissue and breakup of wave fronts during propagation, resulting in fibrillatory or irregularly irregular conduction[24].

Above mentioned factors leads to the development of multiple treatment strategies.

1. Rapid drivers in less quantity were identified in a group of patients with different types of AF using Biatrial phase mapping[25].
2. Ablation lines, maze procedure in atrium interrupts the pathways of Spiral re-entry and multiple wavelets.
3. Continuous biatrial mapping using non-invasive methods gives different results. There is a role mostly for focal sites and multiple wavelets than Rotor Activity[26].

ROLE OF AUTONOMIC NERVOUS SYSTEM:

AF is provoked by parasympathetic and or sympathetic stimuli[27].parasympathetic and or sympathetic activation will lead to provoking of arrhythmias in atrium[28].

Sometimes activation of specific potassium current, $I_{K,\ Ach,}$ leads to shortened atrial duration of action potential and refractoriness heterogeneously, in turn increasing the predisposition to Re-entry. Sympathetic stimuli also sometimes cause a rise in intracellular calcium level causing atriggered activity and automaticity. Near the pulmonary vein-LA junctions and the ligament of Marshall there is some amount of epicardial fat is present in which autonomic

ganglionic plexus is also present, stimulation of this ganglia sometime produces rapid atrial activity.

AF also occurs during conditions of high parasympathetic tone, such as following meals and during sleep, even in persons with structurally and physiologically normal heart called as "vagally mediated AF". During exercise "stimulation of adrenergic system" also provokes AF in some of the patients[29].

Progression from paroxysmal to persistent AF occurs over a period of time, if AF duration is less than 6 months cardio version of AF and maintaining in sinus rhythm will be successful[30]. Studies demonstrate that AF produces structural and electrical remodelling such that "AF begets AF" consistent with progressive nature of atrial fibrillation[31].

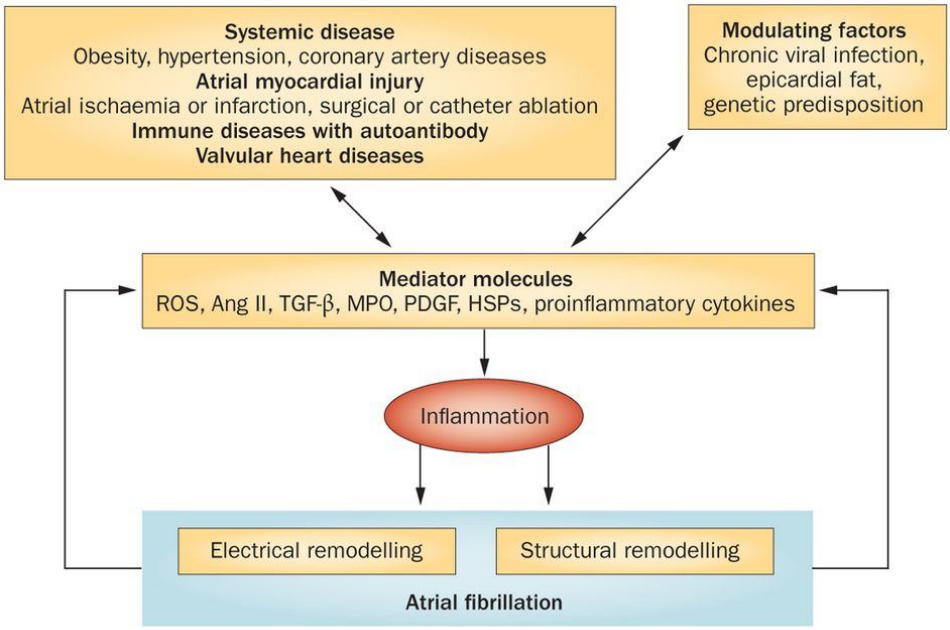


Figure 3: AF Types–promoting remodelling.

Electric remodelling is basically characterized by reduction in duration of action potential duration because of AF and there will be increased risk of delayed after depolarization. Whereas Structural remodelling is mainly due to

death of cells, proliferation of fibroblast and more extracellular matrix (ECM) production which ends up causing fibrosis. This fibrosis formed will prevent electric propagation and favours re-entry. Such interactions between Fibroblast-cardiomyocyte activate re-entry and ectopic impulse production.

RENIN ANGIOTENSIN ALDOSTERONE SYSTEM:

Electrophysiological and structural changes in the atrium and ventricle rises the vulnerability to arrhythmia which are caused by stimulation of renin-angiotensin-aldosterone system[32]. The effects by this RAAS system which are responsible for causing AF are as follows

1. Hemodynamic adverse effects,

2.Increased intracellular calcium from activation of multiple cell signalling cascades,

3. Apoptosis

4. Hypertrophy

5. Oxidative stress

6.Cytokine release and inflammation,

7.Growth related factors that promote fibrosis,

8. Modulation of gap junction and ion channel dynamics.

If there is any variation occurring in the ACE gene expression it ends up in increasing the plasma concentration of angiotensin-II thereby elevating the risk of AF.

Similarly increased in selective expression of ACEs results in dilatation of atrium, fibrosis and increase in vulnerability to AF. Increase in components of renin-angiotensin-aldosterone system synthesized in atrial myocardium during atrial tachypacing is one another mechanism attributed to development of AF.Aldosterone has a stellar part in angiotensin-II mediated inflammation and fibrosis in atria and hence due to this incidence of AF is more in patients with primary hyperaldosteronism.

Previous studies done show that in experimental models of heart failure, Eplerenone and Spiranolactone decreases predisposition to both AF and atrial fibrosis, in these basis patients given eplerenone were associated with decreased occurrence of AF in heart failure patients[33].

INFLAMMATION AND OXIDATIVE STRESS:

These mechanisms also play a role in development of AF which includes, Increase in plasma concentrations of CRP during inflammation linked with development of AF[34]. In case situations like associated with cardiac surgery and pericarditis such mechanisms happen.

2.Interleukin-6 and C-reactive protein are generally raised in patients with AF, so this increased levels of C-reactive protein predicts the development of AF and also it predicts the relapse of AF after cardioversion.

3. If there is variation in Gene expression promoter region of interleukin-6 it may sometime influence the development of AF in post-operative patients.

4. Factors like ageing, inflammation, activation of renin-angiotensin-aldosterone system, Environmental stress also sometimes cause oxidative damage to atrium.

5. When there is upregulation of genes of reactive oxygen species because of oxidative damage it ends up in causing oxidative changes in atrium of patients with AF.

6. There is increase in production of atrial superoxide due to apparent contribution of NAD(P)H oxidase particularly in patients with AF and also in experimental animal models[35]. That's why when we give antioxidant Ascorbate post-operatively it decreases the electrical remodelling and thereby decreased AF[36].

Many researches about genetic forms of AF have been under work for many years but incidence of genetic type of AF is very rare[37], while few population based studies suggest that AF is a heritable disease[38].If there is family history of AF in a first degree relative it will independently increase AF risk by 2 fold[38].Polygenic inheritance is more common than monogenic inheritance in the causation of AF because ion channels are principally affected by monogenic inheritance[39].Genetic linkage studies done in the past have identified the potentially pathogenic loci responsible for development of AF[40].Multiple liability signals are identified at chromosome 4q25 loci[41]. These foci are responsible for expression of transcription factor PITX2; any alteration of this factor is responsible for AF[42].

Familial AF is may be due to several mutations, their role in causation have been studied and identified[43]. These mutations are as follows

1. Gain of function mutation which causes repolarization of potassium currents which in turn leads to a decreased facilitation of atrial re-entry and atrial refractoriness.

2. Many polymorphisms in the causation of AF are idiopathic and they are particularly related with heart diseases having structural anomaly or occurs postoperatively in patients [43].

3.Because of these polymorphisms genes liable forconnexion, sarcolipin, RAAS, eNOS, sodium and potassium channels, interleukin-10, sarcolipin all are affected.

4. The final impact due to this includes changes in conduction, fibrosis, and calcium handling which are the predisposing factors in the development of AF.

CAUSES OF ATRIAL FIBRILLATION:

There are so many causes or risk factors which produces the risk of AF which includes,

CARDIAC

1. Systemic Hypertension

2. Ischemic heart disease

3.Rheumatic mitral valve disease

4.Hypertrophic and dilated cardiomyopathy

5.Congestive cardiac failure

6.Diastolic dysfunction and heart failure

7.Pericarditis and Myocarditis

8.Post cardiac surgery

9.Sick sinus syndrome

10. Atrial septal defect

11.Restrictive cardiomyopathy

12.Mitral valve prolapse syndrome

13.Increased pulse pressure

NON –CARDIAC

1. Smoking
2. Exercise
3. Genetic
4. Familial
5. Amyloidosis
6. Drugs like theophylline
7. Pneumonia
8. Pulmonary embolism
9. Diabetes
10. Age
11. Hyperthyroidism
12. Alcohol intake
13. COPD
14. Obstructive sleep apnoea
15. Pulmonary sleep apnoea

REVERSIBLE OR TEMPORARY CAUSES[44, 45]:-

1. Post cardiac or thoracic surgery
2. Myocardial infarction
3. Pericarditis and Myocarditis
4. Pneumonia
5. Pulmonary embolism
6. Holiday heart syndrome(Binge Alcohol intake)
7. Hyperthyroidism
8. Electrocution.

AF also occurs in some cases of Wolff-Parkinson-white (WPW) syndrome, atrial ectopic tachycardia, AV nodal re-entrant tachycardia and also resolves after catheter ablation therapies for these arrhythmias[46].

ELECTROCARDIOGRAPHIC RISK FACTORS:-

-Left ventricular hypertrophy

ECHOCARDIOGRAPHIC RISK FACTORS:-

-Left atrial enlargement

-Increased LV wall thickness

-Decreased LV fractional shortening

BIOMARKERS:-

-Increased brain natriuretic peptide (BNP)

-Increased C-reactive protein (CRP)

To discuss risk factors in detail foremost is hypertension, most of the patients with AF have systemic hypertension usually with left ventricular

hypertrophy,14% of all cases of AF have associated systemic hypertension[48] as important risk factor .Apart from overt hypertension those patients with even prehypertensive range and wide pulse pressure also have significant risk of developing AF.

Next important risk factor is Coronary heart disease, AF is a frequent problem encountered in acute coronary syndrome[49].There is increased prevalence of both obstructive and non-obstructive CAD in patients with AF than patients without AF.

Similarly valvular heart disease increase the risk of AF with 1.8 to 3.4 times in male and female respectively[48], even though any type of valve lesion can lead to AF left sided valvular heart disease particularly rheumatic heart disease has increased prevalence. Based on many previous studies it was identified that the following lesions has predisposition to AF[50]

Isolated mitral stenosis – 29%

Isolated mitral regurgitation -18 %

Coexisting mitral stenosis and regurgitation – 52%

Mixed mitral and tricuspid regurgitation -70%.

In developing countries rheumatic heart disease is the most common cause of AF that too mitral stenosis is the most common cause of AF, and it is more common in women than men[51]. When compared with western countries Indian patients with RHD develop AF 15- 20 years earlier [52].

One another important risk factor is hypertrophic cardiomyopathy which has been associated with 10% to 28% of AF cases[53].Heart failure and AF often

coexist in such a way that when there is increasing symptoms of heart failure there will be increase in prevalence of AF

The range of increase in prevalence in failure patients based on severity is as follows

NYHA class I = <5% to 10%

NYHA class II-III =10% to 26%

NYHA class IV = 40% to 50%[54].

Cardiac failure increases the risk of AF by 4.5 to 5.9 fold[48].Apart from systolic heart failure sometimes isolated diastolic heart failure also increases the incidence of AF[55].

In congenital heart diseases there is increased prevalence of atrial tachyarrythmias[56]and also it is one of the most common complications in adult patients who presents with congenital heart disease. Among the congenital heart diseases particularly tetrology of fallot, atrial and atrioventricular septal defects, left sided obstructive heart diseases, Ebstein anomaly are associated with increased prevalence of AF , the main reason behind development of AF in all above disease is atrial macro re-entrant arrhythmias . In these lesions left sided hemodynamic changes have animportant role as AF risk determinants[57].

Thyroid dysfunction is strongly associated with development of AF; increased risk of AF is related with overt hyperthyroidism with increased risk around 3 to 6 times than that of persons who are euthyroid. Hence there is linear relationship between AF risk and thyroid function which has been shown by

previous studies. Hence with changes in levels of thyroid stimulating hormone there is increase in relative risk of developing AF which is around

- 1.1 with euthyroid.
- 1.2 times with subclinical hyperthyroid alone
- 1.4 with subclinical hyperthyroid and suppressed TSH

Compared with normal thyroid status[58], in hyperthyroidism it has been hypothesized that increased trigger activity and increased automaticity because *of more* β-adrenergic activity predisposes to AF.

Similarly obesity and obstructive sleep apnoea syndrome both are related with one another and both escalates the risk AF individually[59], rise in systemic inflammatory response and dilatation of atrium are the reason for development of AF in obesity. Some previous studies done showed that for every unit increase in BMI, incident of AF rises by 3% to 7%[60]. Some possible mechanisms postulated in development of AF in sleep apnoea syndrome are

- Hypoxia and autonomic tone surge
- Hypertension.

Diabetes mellitus has been associated with increased risk of 1.4to 1.6 fold, not only because both have similar risk factor profiles like

- Diabetes associated with CAD
- Heart failure
- Obstructive sleep apnoea
- Autonomic dysfunction and
- Systemic inflammation

But also longer duration of disease and poorly controlled glycemic control independently increases the risk AF[61].

Chronic kidney disease is associated with AF frequently and it further increases when associated with common risk factors. Risk of AF increases with severity of renal failure (e.g., with an eGFR of 30-59 and <30ml/min per 1.73 m^2, the increase in relative risk is 1.3-1.6 and 1.6-3.2 times respectively). Similarly end stage renal disease is linked with increased incidence of AF, adjusted relative risk of 1.67-1.77[62].

Regular moderate physical activity usually gives beneficial effects on cardiovascular system and decrease the risk of AF with regular physical activity whereas excessive or vigorous sports activity is linked with increased prevalence of AF[63]. In persons who do such vigorous activity more than three episodes of AF paroxysms are likely to occur in relation with vagal stimulation such as sleep, at rest and postprandial states compared with healthy people[64].

Alcohol consumption and AF relationship has been well known for several years. Acute paroxysmal AF is sometimes associated with binge alcohol intake(Holiday Heart syndrome).Though moderate intake of alcohol does not increase the risk of AF, heavy intake more than or equal to 36g/dl has a higher risk of AF. Also it has been studied that consumption and subsequent withdrawal from alcohol will result in factors like

Impairment of vagal tone,

Hyper adrenergic tone

Changes in conduction properties of atrium all are considered to be predisposes to AF[65].

Similarly smoking has also been related with increased risk of development of AF with more risk in those who are smoking that is with highest percentile of more than 675 cigarette-years)[66]. Sometimes AF recurrence after catheter ablation is linked with regular tobacco use[67].

CLINICAL FEATURES

SYMPTOMS:

Symptoms of AF vary between patients and it ranges from nil symptoms to severe disabling status.

The most common symptoms are,

- Palpitation,
- Fatigue,
- Shortness of breath,
- Chest pain,
- Syncope,

Other less common symptoms are

- Effort intolerance,
- Fluttering or thumping in the chest,
- Giddiness
- Confusion

- Weakness
- Reduced ability to exercise
- Abdominal pain

Patients with paroxysmal AF symptomatic most commonly present with asymptomatic episodes.25% of the patients with AF are almost asymptomatic and they mostly belong to the cadre of patients with persistent AF and elder age group. One uncommon symptom encountered in AF is syncope. Particularly in sick sinus syndrome it can be initiated by long sinus pause on termination of AF whereas in an AF patients with rapid ventricular rate syncope occurs due to either severe fall in blood pressure or because of vasodepressor (neurocardiogenic)syncope that is due to tachycardia induced trigger.

SIGNS:

- Hallmark of AF is an irregularly irregular pulse,
- Pulse deficit: Because of short R-R intervals adequate time for left ventricular filling will not be there which causes low stroke volume and absent peripheral pulse. Hence causing "pulse deficit" in which compared to apical rate peripheral pulse will be less.
- Jugular venous pulsations will be irregular and 'A wave" will be absent
- "First heart sound" is variable.
- Absence of "fourth heart sound".

Sometime the findings are associated with valvular heart disease or myocardial infarction.

CLASSIFICATION OF AF:

They are commonly classified based on the duration of episodes of AF using "simplified scheme revised from the 2006 AF Full revision guideline"[27].The following are the techniques were used to classify atrial fibrillation

- Pacemakers
- Implanted loop recorders
- Defibrillators

By using them we can describe the,

- Rate
- Frequency
- Duration of abnormal atrial rhythms

CLASSIFICATION:

- Paroxysmal,
- Persistent,
- Long standing persistent
- Permanent,
- Non valvular

Table given below has useful clinical relevance, that is we can predict the outcomes of treatment, such as catheter ablation which is better for paroxysmal AF than persistent AF[27],even after restoring to sinus rhythm by cardio version, the duration of the episode(s) of AF is not known, in some patients both paroxysmal and persistent AF can occur together.

Term	Definition
Paroxysmal AF	• AF that terminates spontaneously or with intervention within 7 d of onset. • Episodes may recur with variable frequency.
Persistent AF	• Continuous AF that is sustained >7 d.
Longstanding persistent AF	• Continuous AF of >12 mo duration.
Permanent AF	• Permanent AF is used when there has been a joint decision by the patient and clinician to cease further attempts to restore and/or maintain sinus rhythm. • Acceptance of AF represents a therapeutic attitude on the part of the patient and clinician rather than an inherent pathophysiological attribute of the AF. • Acceptance of AF may change as symptoms, the efficacy of therapeutic interventions, and patient and clinician preferences evolve.
Nonvalvular AF	• AF in the absence of rheumatic mitral stenosis, a mechanical or bioprosthetic heart valve, or mitral valve repair.

"Lone or undetermined AF" is commonly seen in individual's more than 60 years without any structural heart diseases or hypertension.

COMPLICATIONS:-

Some patients because of minimal symptoms or asymptomatic mostly doesn't go for medical advice and their initial presentation itself sometimes present with stroke or thromboembolic complications.

The most common complications are as follows

Heart failure:

Physiologically at rest almost 20% of left ventricular stoke volume is by atrial contraction which is pathologically reduced in AF and additionally it will cause LV dysfunction and irregular rhythm of ventricles[68].AF also decompensate ventricular function. Hence a rise in prevalence of AF is seen in congestive cardiac failure.

Thromboembolism:

One of the most important complications of AF is thromboembolism and it is involved in the causing stroke in elderly patients with AF. The mechanism of thromboembolism is depicted by below image

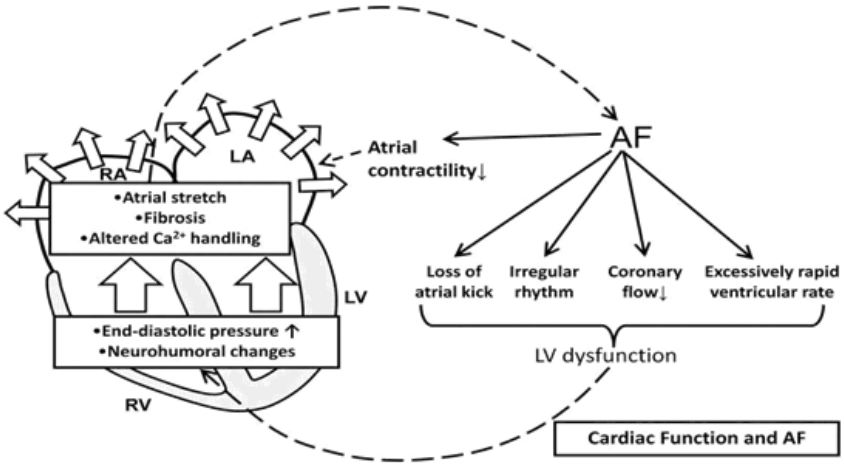


Figure 4: During AF Dynamic interactions between atrial and ventricularfunction.

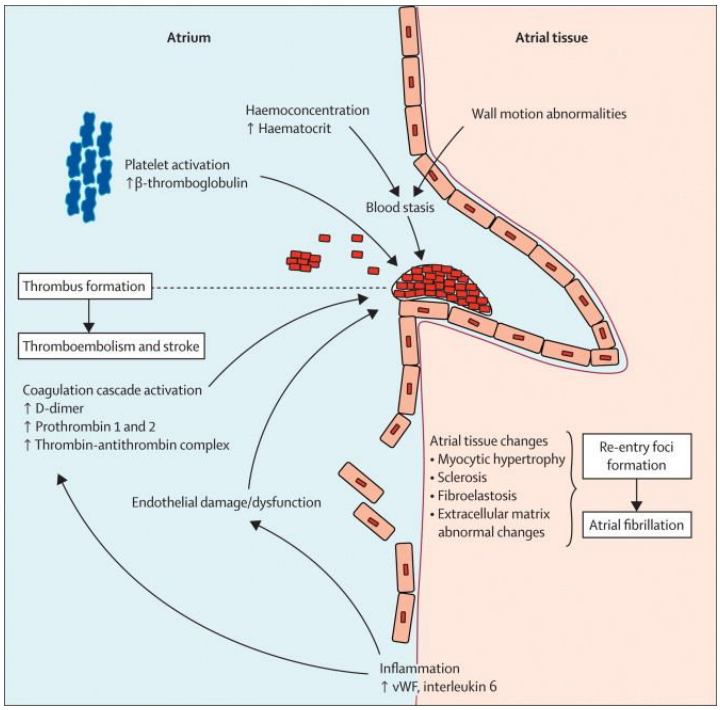


Figure.5:Mechanism of AF related thromboembolism.

For many decades thrombosis-related clinical events have been essentially related to formation of thrombus in the left atrium with subsequent embolization in the cerebral and peripheral circulation. AF satisfies the criteria of Virchow's triad, which are necessary for thrombus formation like blood stasis, endothelial dysfunction and clotting activation. Blood stasis is almost evident in the left atrium of AF patients where flow velocity is significantly reduced alongside impaired contractility of left atrial appendage. It is still unclear, however, if remodelling-related blood stasis is in fact involved in favouring thrombus formation in AF. This hypothesis has been recently challenged by some researchers who demonstrated that atrial remodelling as such does not influence clotting activation and thrombus formation. Endothelial dysfunction is another related component of Virchov's triad which has been seen in patients with AF by measuring several markers of endothelial perturbation such as von Willebrand factor (vWf) and E-selectin. vWf is a glycoprotein secreted by endothelial cells in response to injury and it is usually measured to assess endothelial damage. Several studies consistently showed increased vWf levels in patients with AF. E-selectin is specifically present in endothelial cells and is raised in the blood circulation as a result of endothelial activation. Higher blood levels of E-selectin have been noticed in patients with several types of AF.

Clotting activation is the third component of Virchow's triad which may contribute to thrombosis-related clinical events in AF. Several studies have demonstrated that AF may induce a hyper-coagulation state as shown by

increase of plasma levels of F1+2, D-dimer and fibrinogen. Different authors have investigated the relationship between clotting biomarkers and ischemic events, but results are equivocal; for instance, there are not convincing evidences that fibrinogen is associated with left thrombus or may predict vascular outcomes in AF. A study done earlier showed a significant association between fibrinogen and ischemic stroke suggesting that coagulation system could be implicated in thrombosis-related ischemic events of AF. [18]

3. Stroke:

The most common complication in AF is thromboembolism induced stroke, AF is associated with 5 time higher chance of getting stroke. In a prospective study among patients with stoke, subclinical atrial tachyarrythmias (atrial rate>190 beats /min for 6 minutes) were detected by device interrogation. Hence subclinical atrial tachyarrythmia was independently associated with increased risk of stroke.

Several echocardiographic parameters have been associated with increased risk for stroke in AF, including left ventricular hypertrophy, left ventricular systolic dysfunction, and left atrial enlargement. In a study performed by the SPAF investigators, left ventricular dysfunction on 2-dimensional echocardiography was a strong independent risk factor for thromboembolism.[29] Another prospective study in patients with AF also showed that moderateto-severe left ventricular dysfunction on echocardiography was a strong independent predictor of stroke.[30]Conversely, a subsequent study by the

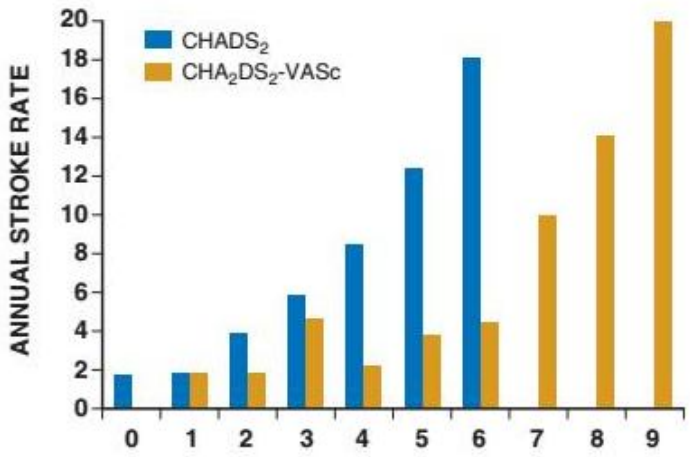
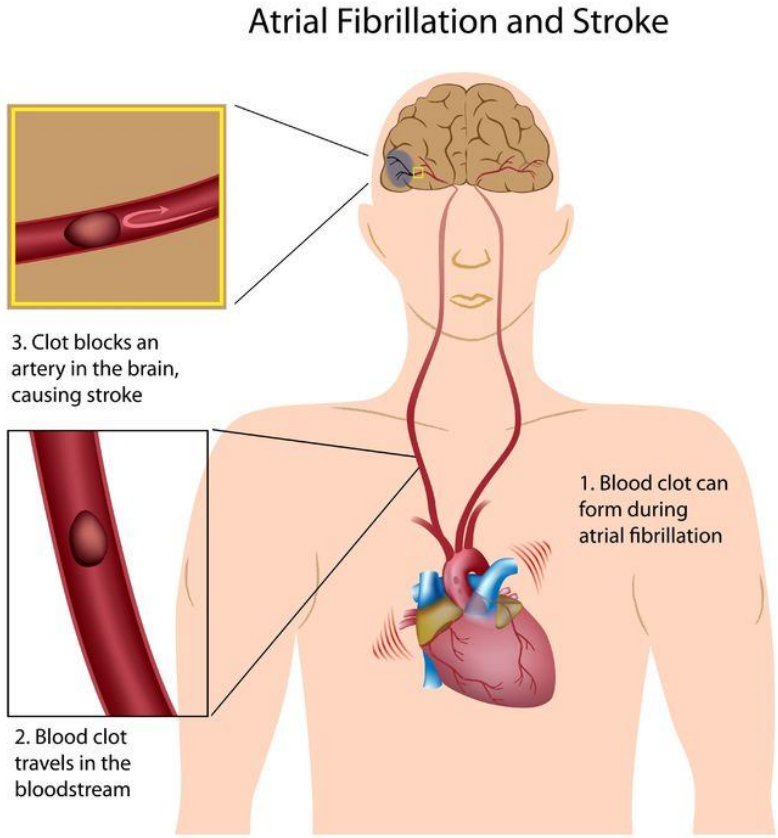


Figure 6. Annual risk for stroke (percent risk per year) based on the $CHADS_2$ and CHA_2DS_2-VASc scores. (Based on data from Lip GY: Implications of the CHA_2DS_2-VASc and HAS-BLED scores for thromboprophylaxis in atrial fibrillation. Am J Med 124:111, 2011.



SPAF investigators failed to identify any echocardiographic parameters that were independently associated with thromboembolism.

Left atrial size measured from the M-mode echocardiogram was shown to be a strong independent predictor of stroke in one study[29] but not in a later study.[30] Left atrial appendage length and width obtained via trans-oesophageal echocardiogram was shown to be associated with risk of thromboembolism on univariate analysis but not multivariate analysis.[31] In the same study, thrombus in the left atrium or left atrial appendage was associated with increased risk of thromboembolism on univariate but not multivariate analysis.[31] Another trans-esophageal echocardiogram study showed that spontaneous echo contrast and complex atherosclerotic plaque in the thoracic aorta were independently predicative of thromboembolism.[18] In summary, the incremental value of echocardiographic parameters for assessing stroke risk in patients with AF is still unproven.

DIAGNOSTIC EVALUATION

Initial diagnostic evaluation of AF includes,

A)Minimum evaluation,

B)Additional investigations, which all depends upon the presentation of patient's clinical situation.

Minimum:

- Patient's clinical history and physical examination findings
- ECG

- Chest X-ray
- Transthoracic Echocardiography
- Blood investigations

Additional investigations:

- 6- minutes walk test
- Exercise testing
- Holter or event monitoring
- Transesophageal echocardiography(TEE)
- Electrophysiological study.

Clinical history and physical examination:

Here we have to assess the following things,

- Nature of the symptomatology
- Clinical types of AF(paroxysmal, persistent, permanent)
- Onset of first episode or day of detection
- Frequency, duration, aggravating factors, modes of initiation and termination of AF
- Response to any drugs administered
- Presence of underlying heart diseases or any reversible conditions

2. Electrocardiogram(ECG):

ECG is the important and essential tool for confirming the diagnosis of AF; the following features are the ECG finding of AF,

- Very irregular and disorganized atrial activity represented as fibrillatory waves called as ‘F’ waves.
- ‘F’ waves may be fine or coarse with varied morphology mistaken for P waves
- Instead of ‘F’ waves, a flat line with irregular R-R intervals may be present.
- Absent or no distinct P waves.
- Atrial rate in AF is ≥ 350 *beats/ minute*
- Ventricular rate is irregularly irregular and it is depends upon number of atrial impulses reaching atrioventricular(AV) node
- Narrow QRS complex unless there is aberrant conduction, preexcitation or bundle branch block.

Fig:ECG with Atrial fibrillation

ECG is used to identify other features in an AF patient to find out other contributing factor or causative disease,

- Rhythm to verify AF
- Morphology and duration of ‘P’waves or ‘F’ waves.
- Bundle branch block, pre-excitation
- Previous MI,

- Other atrial arrhythmias

ECG are helpful in patients who are taking anti arrhythmic therapy, to measure and follow, R-R intervals, QRS complex, QT intervals.

Chest X-ray:

Chest radiograph is done to find out any suspected pulmonary pathology contributing to AF also to identify suspected cardiac failure, to see any cardiac chamber enlargement.

Transthoracic Echocardiography(TTE):

Transthoracic echocardiography (TTE), including two dimensional (2D) imaging and complete Doppler assessment of valves, is recommended for all subjects with AF.[2] TTE allows rapid, safe, relatively comprehensive assessment of cardiac structure and function that can help to define the underlying aetiology of AF and the risk of complications. Recent advances such as harmonic imaging, alone or with micro-bubble contrast agents, allow enhanced endocardial border definition for assessment of left ventricular volumes and function. New modalities such as colour M mode (CMM) and tissue Doppler imaging (TDI) allow more accurate assessment of diastolic function and estimated filling pressures. Assessment of systolic and diastolic left ventricular function in AF may, however, be complicated by irregular RR interval and rapid ventricular rate. Transthoracic imaging usually provides suboptimal visualisation of the atrial appendages and has inadequate sensitivity and specificity for diagnosing LAA thrombus.

Echocardiography hasan important role in management and risk stratification of patients with AF. Echocardiography has also become a fundamental part in the guidelines for management of patients with AF, especially in explaining the mechanisms of systemic thromboembolism in AF.

Transthoracic echocardiography(TTE) allows broad and quick evaluation of cardiac anatomical structure and function. TTE is necessary for the initial evaluation of patient with first episodes of AF and gives evidences to find out the etiology of AF, and helps the physicians to start and decide the treatment approach. There are lot of studies shows that echocardiography helps the physicians in anemergency situation to take decision about antithrombotic prophylaxis. The following can be assessed by TTE.

- Valvular heart disease
- LA and RA size
- LV and RV size and function
- Left ventricular hypertrophy
- LA thrombus(low sensitivity)
- Pericardial disease.

Left ventricular systolic dysfunction identified in TTE in patients with atrial fibrillation can independently predict the risk of stroke(relative risk 2.5; $p<.001$)[69].

LA size is one another important predictor of recurrent AF, large left atrial size is linked with higher risk of developing AF[70], if LA size exceeds >4.5Cm^2 cardioversion is unlikely to be effective[71], LA size >4.0 Cm^2 is a single most strong predictor of increased risk of embolization[72]even in the absence of other causes for increased atrial size, there is increase in size of atria with time in patients of AF[73].

- Chronic duration of AF
- Increased muscle mass
- Left ventricular dilatation.
- Annular calcification
- Mitral regurgitation
- Hypertension

These are the factors which independently influence the LA size. Data from the Framingham study shows that 39% increased risk for subsequent development of AF, if LA dimension increase by 5-mm[74].

Echocardiography is also useful in evaluating the atrial function. Both LA compliance and volume reduction has been noted with the onset of AF and it also reduces cardiac function and in turn there will be increase in risk of thromboembolism.

Similarly in patients with sinus rhythm, presence of severe LV diastolic failure is linked with a higher risk for AF and heart failure, as evaluated by ECHO. Septal thickness(IVSD) and left ventricular posterior wall thickness (LVPWD) are other independent factors influencing the prognosis of AF.

They are also useful in predicting thromboembolism based on below features

- Valve disease
- Left ventricular systolic dysfunction
- Left atrial dilatation
- Complex aortic atheroma
- Left atrial appendage thrombus > sludge > smoke
- Reduced LAA velocities (< 20 cm/s)

Blood investigations:

In a patient with first episode of AF and in those whom ventricular rate is difficult to control following test are required

Thyroid function test

Liver Function test

Renal function test.

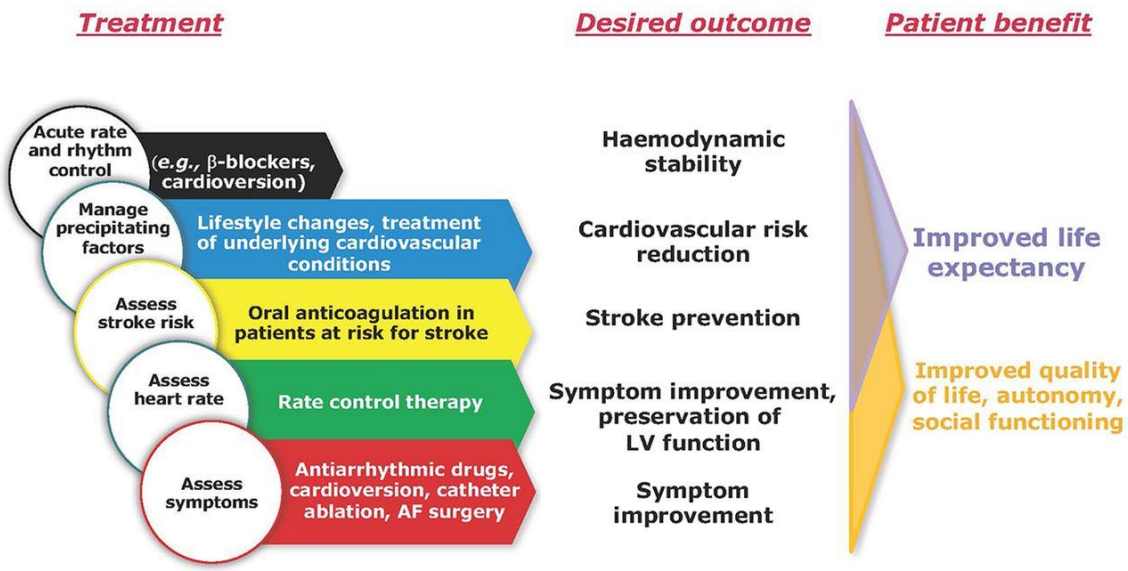
Additional investigations:

One are more of the following investigations may be required depending upon the clinical situations.

1. 6-min walk test	• If the adequacy of rate control is in question
2. Exercise testing	• If the adequacy of rate control is in question
	• To reproduce exercise-induced AF
	• To exclude ischemia before treatment of selected patients with a type IC antiarrhythmic drug
3. Holter or event monitoring	• If diagnosis of the type of arrhythmia is in question
	• As a means of evaluating rate control
4. TEE	• To identify LA thrombus (in the LAA)
	• To guide cardioversion
5. Electrophysiological study	• To clarify the mechanism of wide-QRS-complex tachycardia
	• To identify a predisposing arrhythmia such as atrial flutter or paroxysmal supraventricular tachycardia
	• To seek sites for curative AF ablation or AV conduction block/modification

TREATMENT

The basic algorithm of management in atrial fibrillation is depicted below



Acute management:

New onset AF: It presents with severe hypotension, pulmonary edema, or angina should be electrically cardioverted starting with aQRS synchronous shock of 200 J, ideally after sedation or anaesthesiais achieved. Greater shock energy and different electrode placements may be tried if the shock fails to terminate AF. If AF terminates and reinitiates, administration of an antiarrhythmic drug, such as ibutilide,and repeat cardio version may be considered. If the patient is stable,immediate management involves rate control to alleviate or prevent symptoms, anticoagulation if appropriate, and cardio version to restore sinus rhythm if AF is persistent.

Anticoagulation strategies for newonset AF are debated. In the absence of contraindications, it is usually appropriate to initiate systemic anticoagulation with heparin immediately, while evaluation and other therapies are implemented.

Acute rate control can be achieved with beta blockers, calcium channel blockers such as oral or i.v verapamil and diltiazem, Digoxin is added in patient with cardiac failure patients, because it does not have negative inotropic effects, particularly if use of AV nodal–blocking agents is limited by poor tolerance or is contraindicated. Its effect is modest but synergistic with the other AV nodal–blocking agents, but it is particularly limited when sympathetic tone is elevated. Typically, the goal of acute rate control is to reduce the ventricular rate to less than 100/min, but the goal must be guided by the clinical situation.

Long term management of atrial fibrillation:

1. Pharmacological rate control:

According to ACC/AHA guidelines the following table describe about the pharmacological rate control measures.

CLASS	INDICATION
Class I (indicated)	Measurement of the heart rate at rest and control of the rate with pharmacologic agents (in most cases either a beta blocker or nondihydropyridine calcium channel antagonist) are recommended for patients with persistent or permanent AF In the absence of preexcitation, intravenous administration of beta blockers (esmolol, metoprolol, or propranolol) or nondihydropyridine calcium channel antagonists (verapamil, diltiazem) is recommended to slow the ventricular response to AF in the acute setting, with caution being exercised in patients with hypotension or heart failure Intravenous administration of digoxin or amiodarone is recommended to control the heart rate in patients with AF and heart failure who do not have an accessory pathway In patients who experience symptoms related to AF during activity, the adequacy of heart rate control should be assessed during exercise, with pharmacologic treatment being adjusted as necessary to keep the rate in the physiologic range Digoxin is effective after oral administration to control the heart rate at rest in patients with AF and is indicated for patients with heart failure or left ventricular dysfunction and for sedentary individuals
Class IIa (reasonable)	A combination of digoxin and either a beta blocker or nondihydropyridine calcium channel antagonist is reasonable to control the heart rate both at rest and during exercise in patients with AF. The choice of medication should be individualized and the dose modulated to avoid bradycardia It is reasonable to use ablation of the AV node or accessory pathway to control the heart rate when pharmacologic therapy is insufficient or associated with side effects Intravenous amiodarone can be useful to control heart rate in patients with AF when other measures are unsuccessful or contraindicated When electrical cardioversion is not necessary in patients with AF and an accessory pathway, intravenous procainamide or ibutilide is a reasonable alternative
Class IIb (may be considered)	When the ventricular rate cannot be adequately controlled both at rest and during exercise in patients with AF by a beta blocker, nondihydropyridine calcium channel antagonist, or digoxin, alone or in combination, oral amiodarone may be administered to control the heart rate Intravenous procainamide, disopyramide, ibutilide, or amiodarone may be considered for hemodynamically stable patients with AF involving conduction over an accessory pathway When the rate cannot be controlled with pharmacologic agents or tachycardia-mediated cardiomyopathy is suspected, catheter-directed ablation of the AV node may be considered in patients with AF to control the heart rate
Class III (not indicated)	Strict rate control (<80 beats/min at rest or <110 beats/min during a 6-minute walk) is not more beneficial than a resting rate of <110 beats/min in asymptomatic patients with persistent AF and an ejection fraction >40%, although uncontrolled tachycardia can lead to reversible left ventricular dysfunction over time Digitalis should not be used as the sole agent to control the rate of ventricular response in patients with paroxysmal AF Catheter ablation of the AV node should not be attempted without a prior trial of medication to control the ventricular rate in patients with AF In patients with decompensated heart failure and AF, intravenous administration of a nondihydropyridine calcium channel antagonist may exacerbate the hemodynamic compromise and is not recommended Intravenous administration of digitalis glycosides or nondihydropyridine calcium channel antagonists to patients with AF and a preexcitation syndrome may paradoxically accelerate the ventricular response and is not recommended

2. Rhythm control:

a) Cardioversion

First line drugs recommended for pharmacological cardio version includes the following drugs,

- Flecainide,
- Dofetilide ,
- Propafenone ,
- Ibutilide.

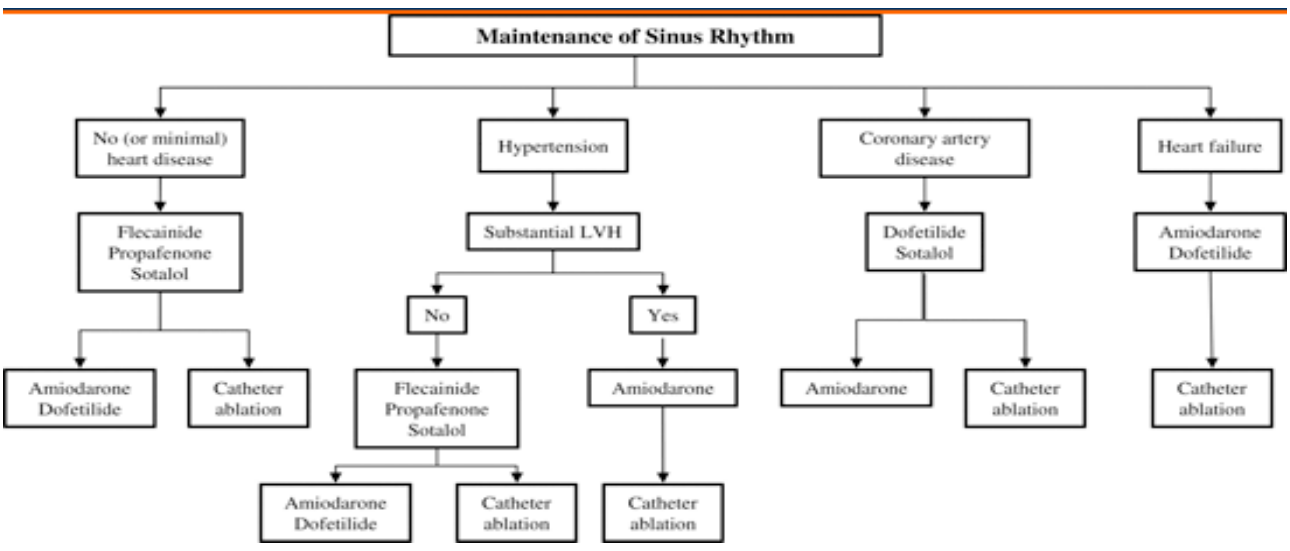
b) Direct – current (DC) cardio version;

Recommended when the ventricular rate is rapid and does not respond quickly to drug therapy in patients with

- Ischemic heart disease,
- Hypotension,
- Cardiac failure,
- WPW syndrome,
- Rapid ventricular rate,
- Patients with hemodynamic instability.

c)Maintenance of sinus rhythm:

According to ACC/AHA guidelines the following table describe about the maintenance of sinus rhythm measures.



B)Special situations:

Post-operative Atrial fibrillation:

Prophylactic treatment with beta blocker to prevent post-operative AF in patients planned for cardiac surgery.

Acute Myocardial Infarction:

Electrical cardio version is recommended for patients with hemodynamic compromises or ongoing ischemia or when drugs failed to control the rate.

AF in WPW syndrome:

Catheter ablation of the accessory pathway is recommended for patients with symptomatic AF in WPW syndrome.

Hyperthyroidism:

Beta blockers are the first line drugs for rate control in thyrotoxicosis patients with AF.

Hypertrophic Cardio myopathy:

The drugs used preferably either disopyramide with beta blocker,verapamil, or diltiazam or amiodarone for rate control.

AF during Pregnancy:

Recommended drugs are dioxin, beta blocker or a nondihydropyridine calcium channel antagonist for rate control. DC cardio version is recommended in hemodynamically unstable patients.

Pulmonary Disease:

Verapamil or diltiazam is used for rate control in patient with COPD.

Prevention of Thromboembolic Complications:

The main aim of treatment in patients with AF is to prevent thromboembolic complications such as stroke. There are two scoring systems have developed for 'risk stratification' they are as follows,

1. CHADS2scoring system:

A simple method to "risk stratify" patients is based on "$CHADS_2$ score" By using this scoring system we can predict the direct relationship between $CHADS_2$ score and the annual risk of stroke in patients with AF in the absence of warfarin or aspirin treatment, this score is very simple and it has predictive value.

2. CHA2DS2-VASc scoring system:

But recent studies have shown that" CHA_2DS_2–VASc score" more accurately differentiates low risk from intermediate risk patients.

$CHADS_2$ -> CHA_2DS_2VASc

CHADS2 Risk	Score
CHF	1
Hypertension	1
Age > 75	1
Diabetes	1
Stroke or TIA	2

CHA2DS2-VASc Risk	Score
CHF or LVEF ≤ 40%	1
Hypertension	1
Age ≥ 75	2
Diabetes	1
Stroke/TIA/ Thromboembolism	2
Vascular Disease	1
Age 65 - 74	1
Female	1

From ESC AF Guidelines
http://escardio.org/guidelines-surveys/esc-guidelines/GuidelinesDocuments/guidelines-afib-FT.pdf

Based on the scores the therapies are recommended as follows,

Aspirin is used prophylatically when" CHA_2DS_2–VASc score" of 0.

Aspirin for stroke prevention is recommended for patient with 'CHADS2score 'of0.

3. Aspirin and or oral anticoagulant,When the $CHADS_2$ score is 1.

Newer Anticoagulants:

Dabigatran, Rivaroxaban and apixaban for patients with AF in whom maintenance of therapeutic INR during treatment with warfarin is difficult.

Low molecular heparin is an alternative to unfractionated heparin for initiation of anticoagulation with warfarin.

Surgical removal or closure of LA appendage.

"HAS –BLED scoring system" was developed to risk stratify patients susceptible for bleeding complications. As the score increases from 0 to maximum of 9, there is a gradual increase in bleeding in patients treated with warfarin.

HAS-BLED

Letter	Clinical Characteristic	Points
H	Hypertension	1
A	Abnormal Liver or Renal Function	1 or 2
S	Stroke	1
B	Bleeding	1
L	Labile INR	1
E	Elderly (age > 65)	1
D	Drugs or Alcohol	1 or 2
Maximum Score		9

D) Non pharmacologic management of AF:

The following are the non-pharmacological options available for management of AF,

- Pacing,
- Catheter ablation,
- Surgical removal or closure of LA appendage,
- Maze procedure.

Atrioventricular junction ablation with pacing is an effective strategy for a small number of highly symptomatic patients with AF, but it is highly invasive and creates dependence on an implanted pacemaker. It is commonly considered in patients with AF in whom neither sinus rhythm nor rate control can be achieved nor in patients who already have a pacemaker for bradycardia-tachycardia syndrome. Several studies have shown improved quality of life and improvement in ventricular function with this strategy. In patients with symptomatic heart failure and permanent AF, biventricular pacing can also be useful with atrioventricular junction ablation.

Various pacing strategies, including multi-site atrial pacing, atrial anti-tachycardia pacing, and atrial defibrillator therapy, have been tested for the prevention and treatment of AF, but no studies have convincingly shown a significant benefit for these patients.

In patients with primary typical right atrial flutter which degenerates into AF, a "hybrid" procedure involving atrial flutter ablation and treatment with an

antiarrhythmic drug may be an effective strategy. However, this population represents only a small minority of AF patients.

The surgical Maze procedure was developed nearly 2 decades ago, and clinical results have been quite good, with reported success rates of over 90% in many trials. However, the requirement for open-heart surgery has necessarily restricted this therapy to patients undergoing cardiac surgery for other indications.

JUSTIFICATION OF STUDY

- Atrial fibrillation(AF) is the most common sustained cardiac arrhythmia in clinical practice and increasing in prevalence
- Atrial tachyarrhythmia characterized by predominantly uncoordinated atrial activation with consequent deterioration of atrial mechanical function
- Prevalence roughly doubles with each advancing decade
- This study is intended to find out the varied presenting symptoms of AF and also possible underlying predisposing factors- cardiac and non-cardiac in Indian context
- If identified early and treated properly, AF rarely causes serious or life-threatening complications

MATERIALS AND METHODS

Study design: Cross-sectional study

Study period: One year

Study area: Govt. Kanyakumari Medical College and Hospital.

Study population: Patients with New onset Atrial Fibrillation (in patients and out patients) in Department of General medicine and Department of Cardiology, Govt. Kanyakumari Medical College and Hospital.

Sample Size: Was *calculated based on α level 5%[95% confidence interval],* absolute accuracy at 9%, so by calculating by this sample size is 50.since inflow of patients with AF to our hospital is very high, I have taken up sample size as 100 to my study.

Ethical clearance: Ethics committee clearance was obtained.

Consent: Informed consent obtained from all subjects for clinical examination and for doing investigations. Patient confidentiality maintained.

Inclusion criteria: Patients aged more than 18yrs, Patients with clinically and electrocardiographically proven atrial fibrillation and hemodynamically stable patients.

Exclusion criteria: Patients with atrial arrhythmias other than atrial fibrillation and hemodynamically unstable patients.

Methodology:

100 patients with Atrial fibrillation were analysed in this study, and their general and clinical data was included in the proforma.

Patient’s age, sex, clinical symptoms and past history of Systemic Hypertension, Rheumatic heart disease, Coronary Artery Disease, chronic obstructive pulmonary disease, Hyperthyroidism, Cardiomyopathy, Congenital heart disease, Stroke, and treatment history, were taken in to account.

Diagnosis of atrial fibrillation was done by absent P waves, fibrillatorywaves, irregularly irregular ventricular rate in ECG were taken as the evidence for AF. Evaluation regarding etiology of AF is done by using ECG and Transthoracic echocardiogram, Chest radiograph were done in all patients.

Diagnosis of Systemic hypertension was made by blood pressure with systolic BP > 140 mmHG and /or Diastolic BP > 90mmHG.

Presence of ‘T’ wave inversion and Significant Q waves in ECG, Regional wall motion abnormality in ECHO were taken as evidences for coronary artery disease.COPD was diagnosed by using history of chronic cough and history of smoking, Chest radiograph, ECG, Transthoracic ECHO.

Thyroid Function tests were done only for ‘at risk’ cases and those who are presenting with signs and symptoms of hyperthyroidism. History of smoking and Alcohol were asked in all patients.

Clinical examination:

- General examination
- Vitals:-
- Pulse rate,
- Pulse deficit,
- Blood pressure,

- Systemic examination,
- Signs of hyperthyroidism,

Laboratory data:-

- Conventional 12 lead ECG with rhythm strip,
- Transthoracic echocardiographic examination,
- Complete blood count,
- .Renal function test
- Lipid profile Blood glucose levels-fasting and post prandial,
- Chest Radiograph,
- Thyroid function tests in selected patients,

In our study, All patients were analyzed with 2D ECHO, M MODE and Colour Doppler to find out the coronary heart diseases, if structural heart disease like congenital heart diseases, hypertensive heart disease, and dilated cardiomyopathies, hypertrophic cardiomyopathy.

Transthoracic echocardiographic assessment also includes the search for the presence of left atrial clot.

Transthoracic echocardiography (TTE) was done in all patients and the following parameters were assessed.

LVIDd(cm)

Ejection fraction%

5.RWMA

6.Valvular lesions

7.RA and LA Size(cm)

8.LA Clot

9.RV systolic dysfunction

Analysis of Data:

The different presentations of AF will be provided as % with 95% confidence interval.

The different predisposing factors will be provided as %.

The different parameters of Transthoracic Echocardiography will be provided in %.

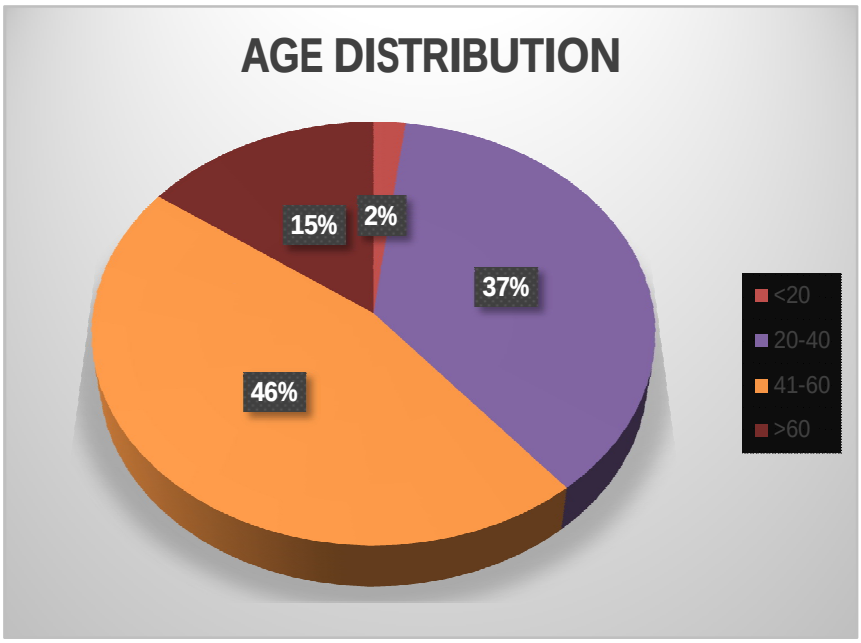
The collected data were analysed with IBM.SPSS statistics software 21.0 Version. To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean and S.D were used for continuous variables. To find the significant difference between the bivariate samples in Independent groups the unpaired sample t-test was used.To find the significance in categorical data Chi-Square test was used. In all the above statistical tools the probability value <.05 is considered as significant level.

RESULTS

Table 1: Age distribution

AGE IN YEARS	NO OF PATIENTS	PERCENTAGE
<20	2	2%
20-40	37	37%
41-60	46	46%
>60	15	15%

Chart 1: Age Distribution

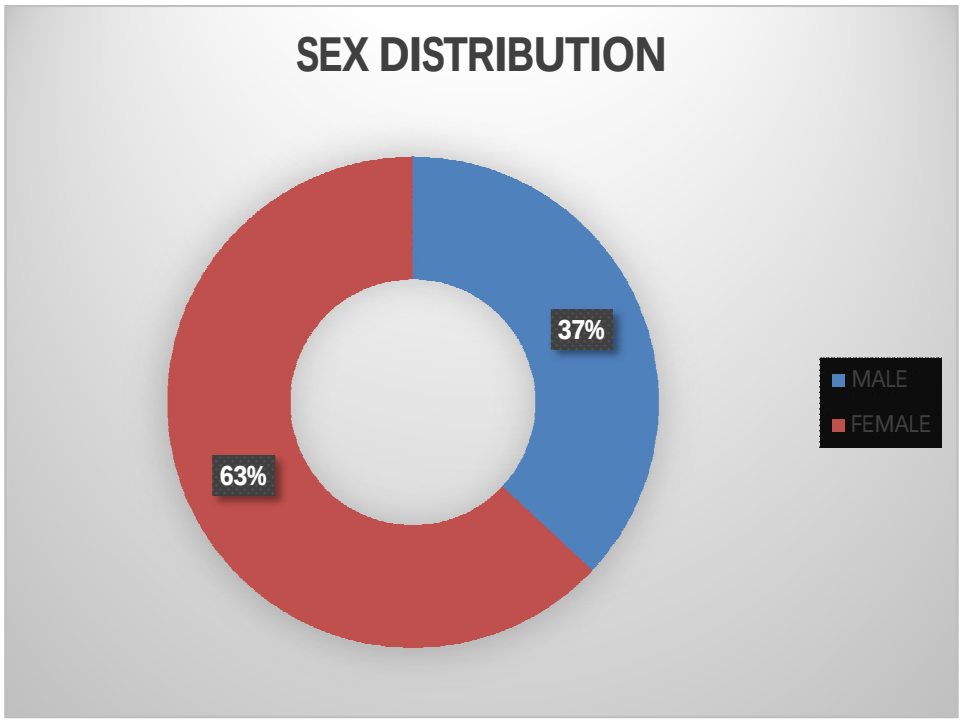


Among our study group most commonly patients were in 41-60 age group followed by 20-40 age group.

Table 2: Sex distribution

SEX DISTRIBUTION	NO OF PATIENTS	PERCENTAGE
MALE	37	37%
FEMALE	63	63%

Chart 2: Sex distribution

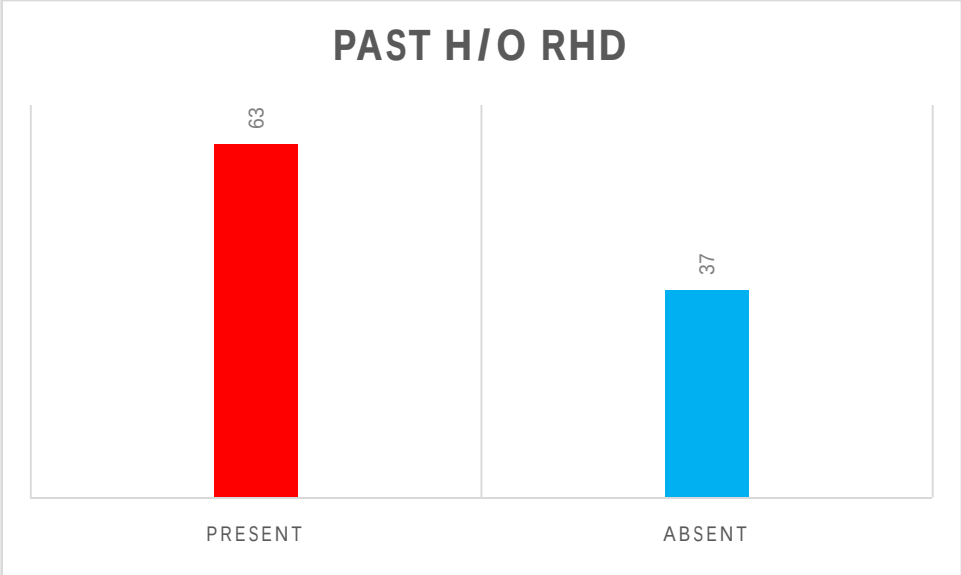


Females were commonly seen in our study group more than males with a male female ratio of 1:1.7.

Table 3: Past history of rheumatic heart disease

PAST H/O RHD	NO OF PATIENTS	PERCENTAGE
PRESENT	63	63%
ABSENT	37	37%

Chart 3: Past history of rheumatic heart disease

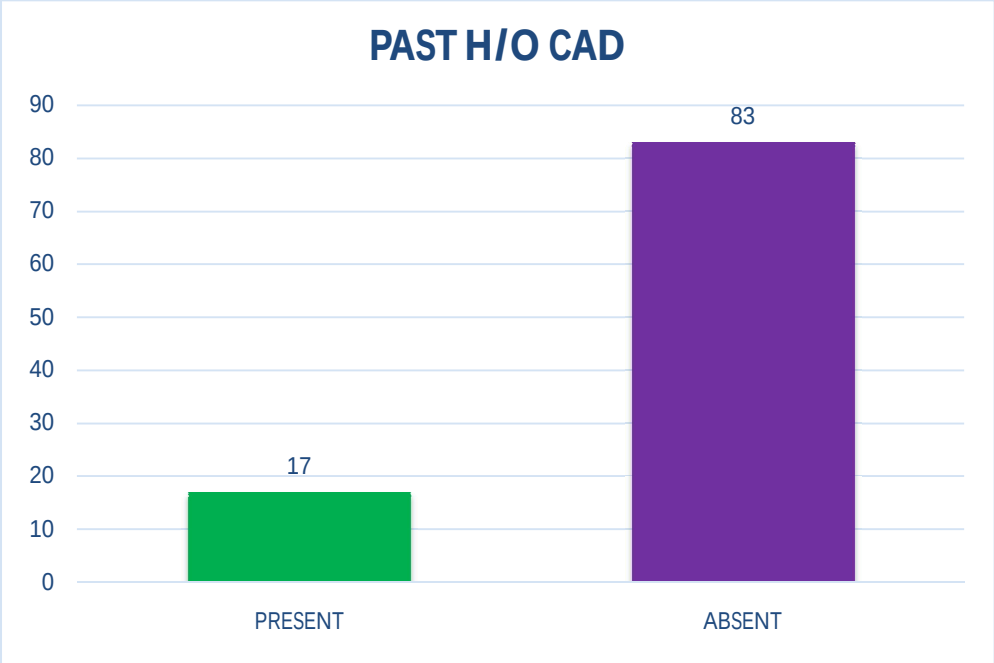


Among our study group around 63 patients had past history of rheumatic heart disease which one of most common etiology

Table 4: Past history of coronary artery disease

PAST H/O CAD	NO OF PATIENTS	PERCENTAGE
PRESENT	17	17%
ABSENT	83	83%

Chart 4: Past history of coronary artery disease

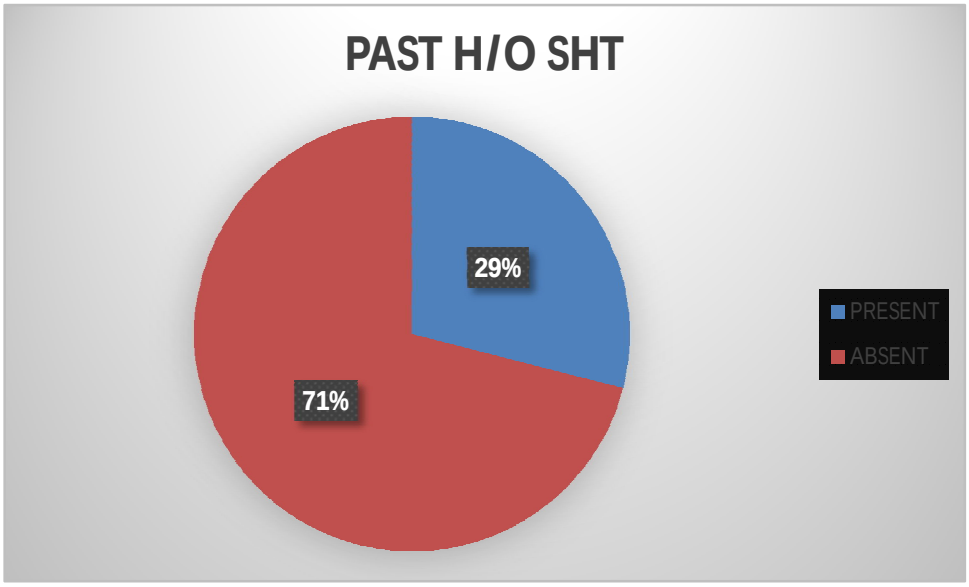


Among our study group around 17 patients had past history of coronary artery disease

Table 5: Past history of systemic hypertension

PAST H/O SHT	NO OF PATIENTS	PERCENTAGE
PRESENT	29	29%
ABSENT	71	71%

` Chart 5: Past history of rheumatic heart disease

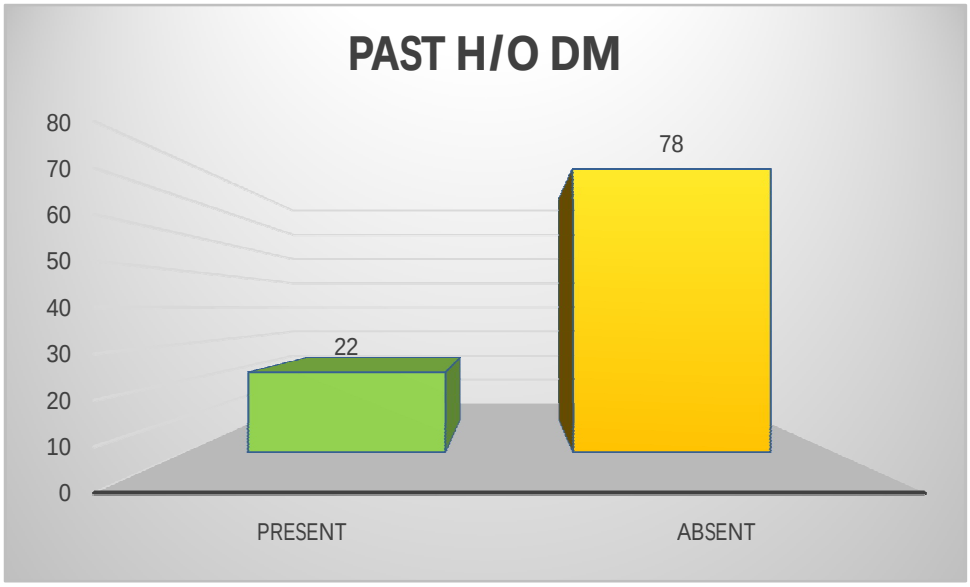


Among our study group around 29patients had past history of systemic hypertension

Table 6: Past history of diabetes mellitus

PAST H/O DM	NO OF PATIENTS	PERCENTAGE
PRESENT	22	22%
ABSENT	78	78%

Chart 6: Past history of diabetes mellitus

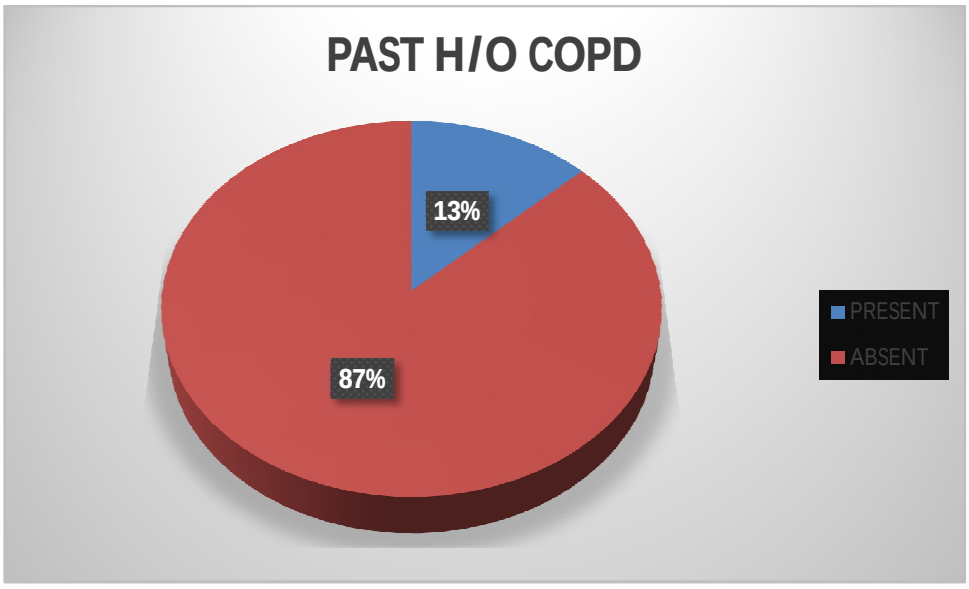


Among our study group around 22 patients had past history of diabetes mellitus.

Table 7: Past history of COPD

PAST H/O COPD	NO OF PATIENTS	PERCENTAGE
PRESENT	13	13%
ABSENT	87	87%

Chart 7: Past history of COPD

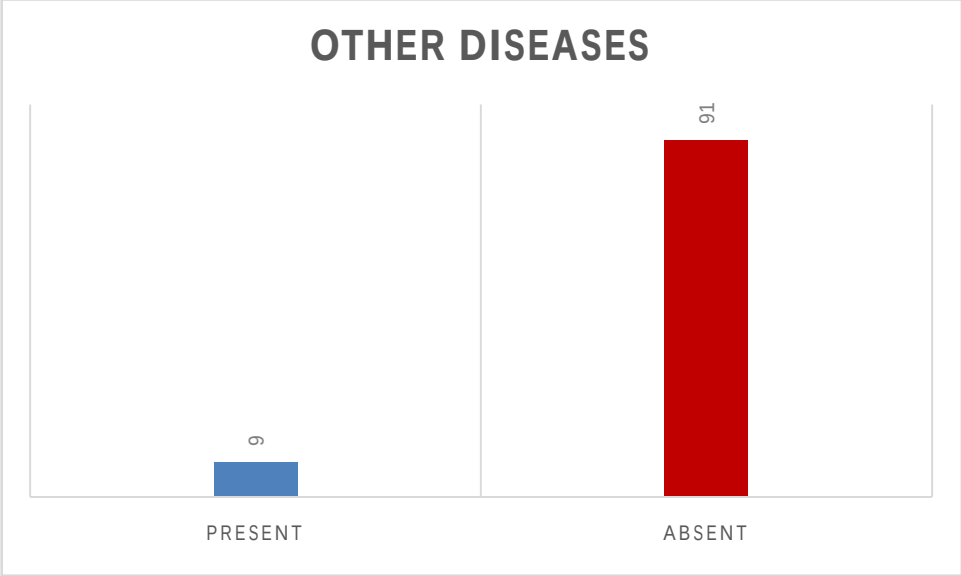


Among our study group around 13 patients had past history of chronic obstructive pulmonary disease.

Table 8: Other associated diseases

OTHERS	NO OF PATIENTS	PERCENTAGE
PRESENT	9	9%
ABSENT	91	91%

Chart 8: Other associated diseases



Among our study group around 9 patients had history of other diseases.

Chart 9: Other associated diseases

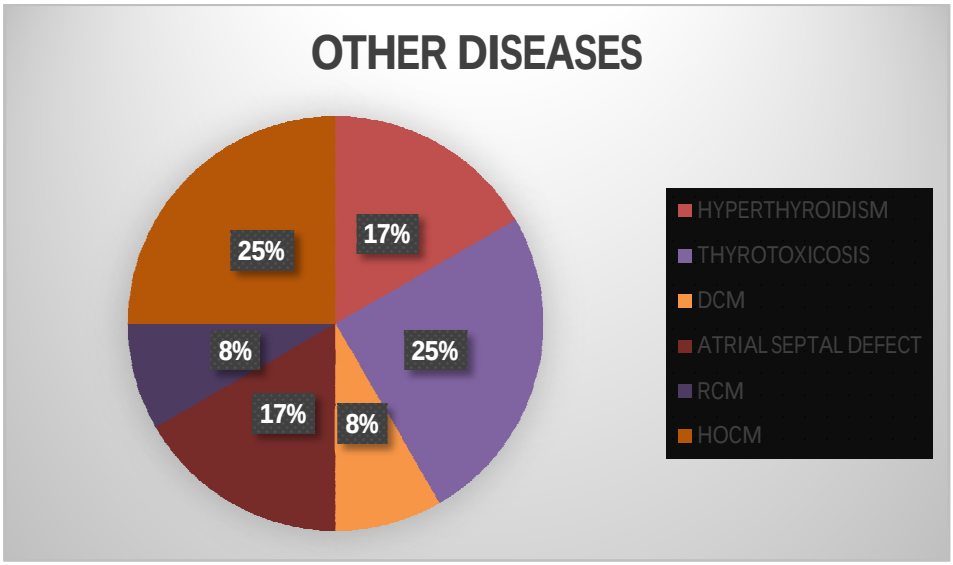


Table 9: Other associated diseases

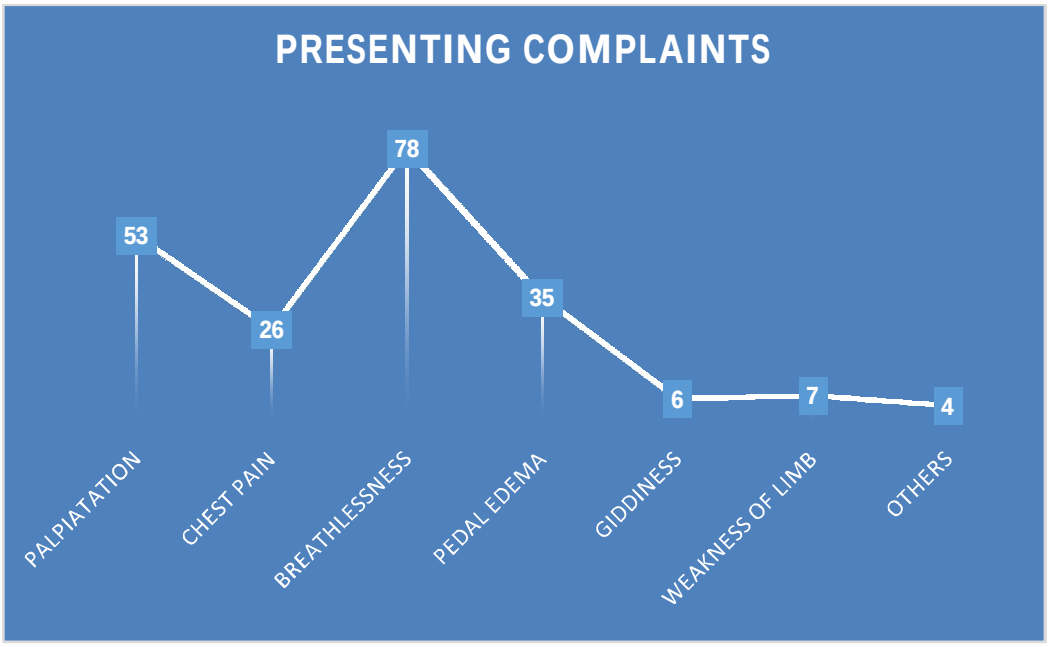
OTHER DISEASES	NO OF PATIENTS	PERCENTAGE
HYPERTHYROIDISM	2	17%
THYROTOXICOSIS	3	25%
DCM	1	8%
ATRIAL SEPTAL DEFECT	2	17%
RCM	1	8%
HOCM	3	25

Among our study group patients had other diseases like thyroid disorders, cardiomyopathies and ASD.

Table 10: Presenting complaints

PRESENTING COMPLAINTS	NO OF PATIENTS	PERCENTAGE
PALPIATATION	53	53%
CHEST PAIN	26	26%
BREATHLESSNESS	78	78%
PEDAL EDEMA	35	35%
GIDDINESS	6	6%
WEAKNESS OF LIMB	7	7%
OTHERS	4	4%

Chart 10: Presenting complaints



Among our study group most common presenting complaints is breathlessness followed by palpitation. Chest pain is seen in 26 patients and pedal edema in 35 patients.

Chart 11: Number of complaints

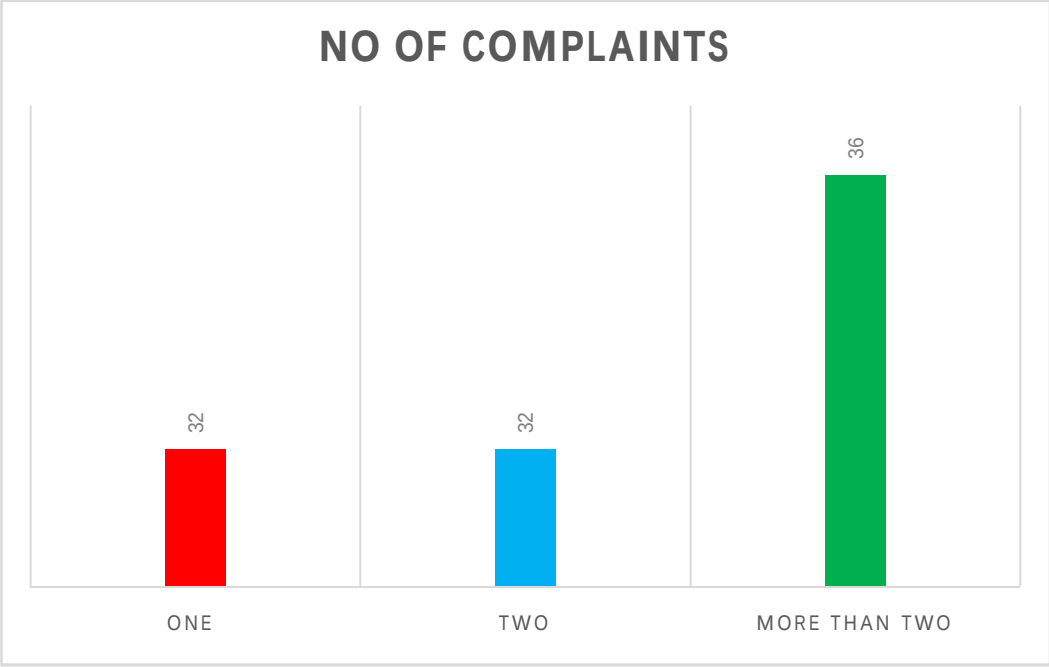


Table 11: Number of complaints

NO OF COMPLAINTS	NO OF PATIENTS	PERCENTAGE
ONE	32	53%
TWO	32	26%
MORE THAN TWO	36	78%

In our study patients 32 patient's presented with single complaints and 32 patients with two complaints where as rest of patients presented with more than 2 complaints.

In our study group 25 patients had some sort of personal habits like smoking and alcohol intake.16 patients had smoking habit and 19 patients had habit of alcohol intake.

Chart 13: Smoking habit

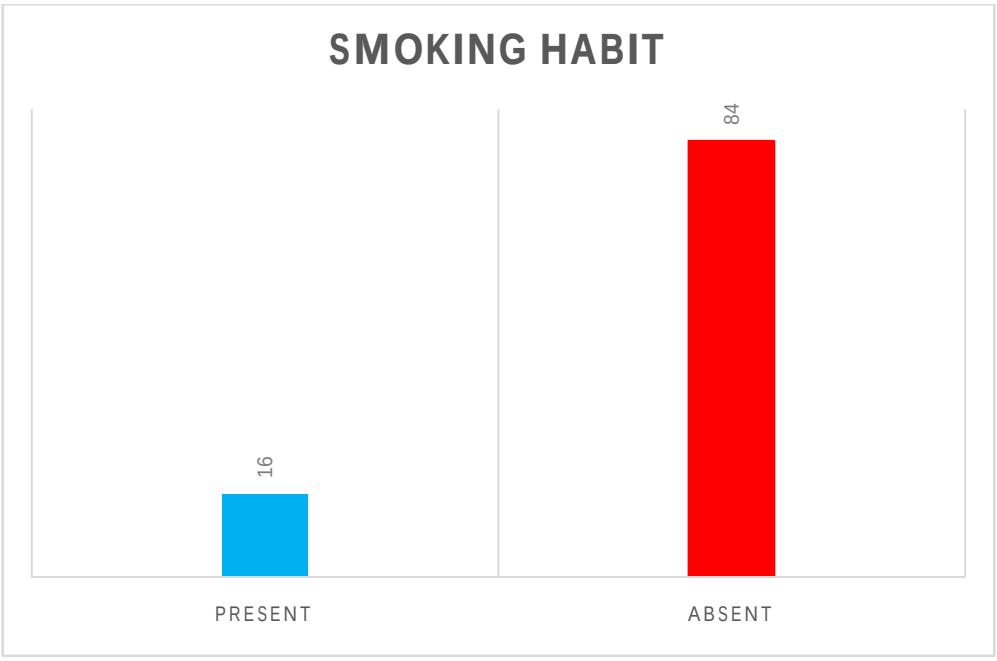


Table 13: Smoking habit

SMOKING HABIT	NO OF PATIENTS	PERCENTAGE
PRESENT	16	16%
ABSENT	84	84%

Table 14: Alcoholic habit

ALCOHOLIC	NO OF PATIENTS	PERCENTAGE
YES	19	19%
NO	81	81%

Chart 14: Alcoholic habit

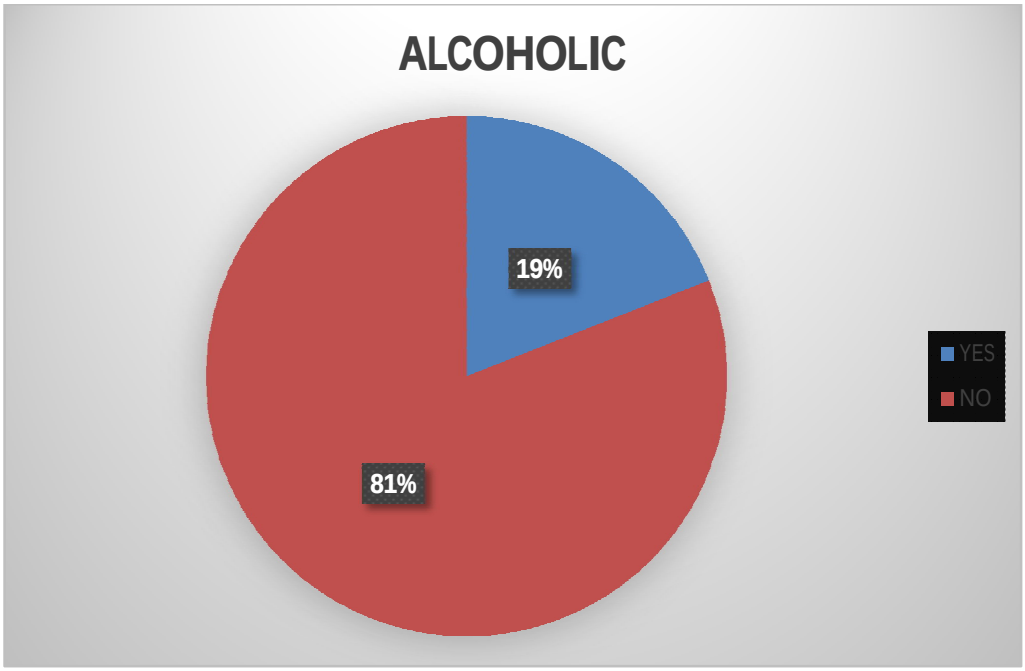
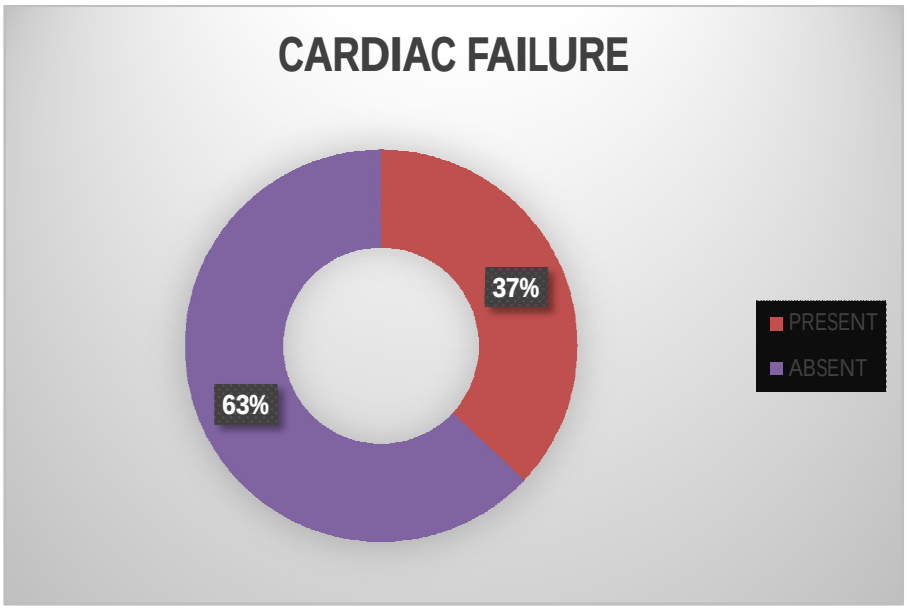


Table 15: Cardiac failure

CARDIAC FAILURE	NO OF PATIENTS	PERCENTAGE
PRESENT	37	37%
ABSENT	63	63%

Chart 15: Cardiac failure



In our study patients around 37 patients had symptoms of cardiac failure with different ranges of ejection fraction.

Chart 16: Heart rate

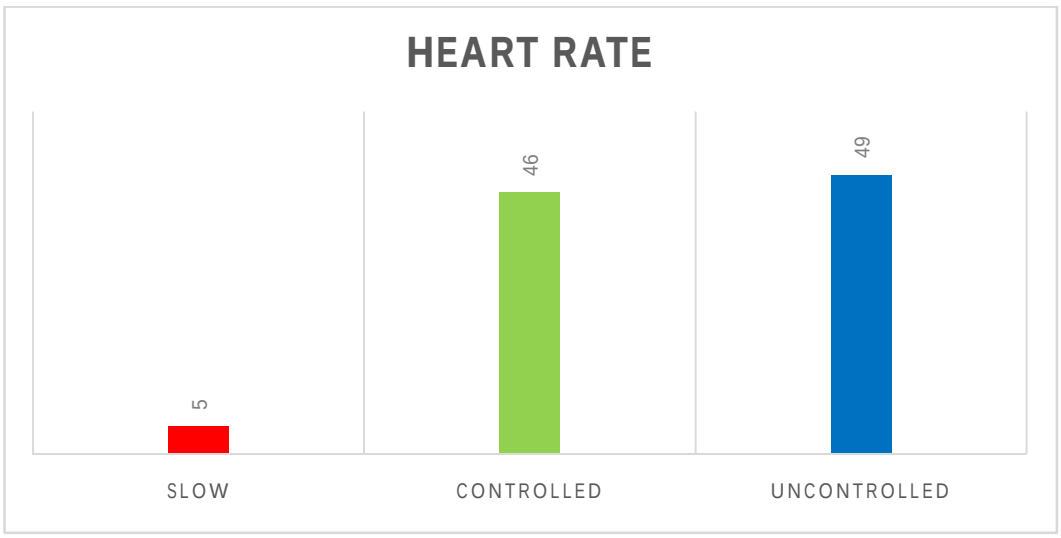


Table 16: Heart rate

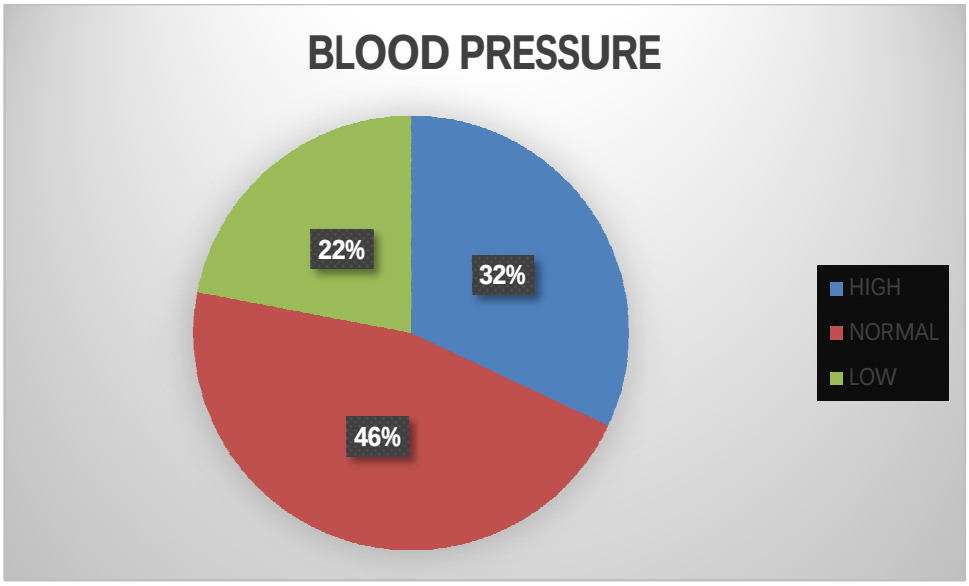
HEART RATE	NO OF PATIENTS	PERCENTAGE
SLOW	5	5%
CONTROLLED	46	46%
UNCONTROLLED	49	49%

Around half of patients in our study (n=49) uncontrolled heart rate or tachycardia while rest had normal rate and only two patients had bradycardia.

Table 17: Blood pressure

BLOOD PRESSURE	NO OF PATIENTS	PERCENTAGE
HIGH	32	32%
NORMAL	46	46%
LOW	22	22%

Chart 17: Blood pressure



Abnormal blood pressure either high (n=32) or low (n=22) is seen in 54 patients while the rest had normal blood pressure.

Chart 18: Left Ventricular Internal Dimension-Diastole.

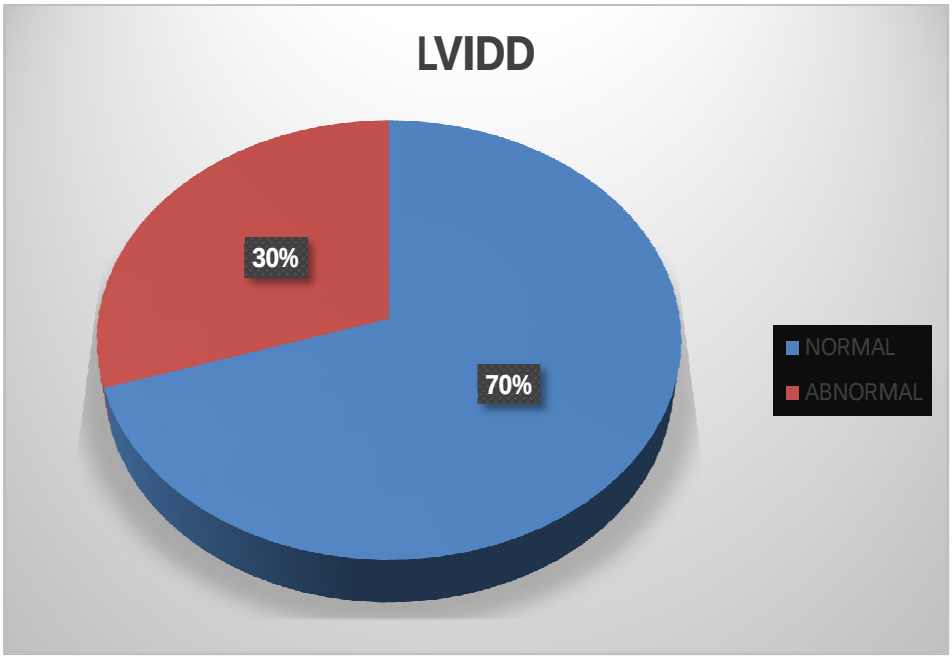


Table 18: Left Ventricular Internal Dimension-Diastole.

LVIDd	NO OF PATIENTS	PERCENTAGE
NORMAL	70	70%
ABNORMAL	30	30%

In our study group Left ventricular internal dimension during diastole was abnormal or increased in 30 patients among which the increase was mild in 22 patients and moderate in 8 patients.

Table 19: Abnormal Left Ventricular Internal Dimension-Diastole.

ABNORMAL LVIDd	NO OF PATIENTS	PERCENTAGE
MILD	22	73%
MODERATE	8	27%

Chart 19: Abnormal Left Ventricular Internal Dimension-Diastole

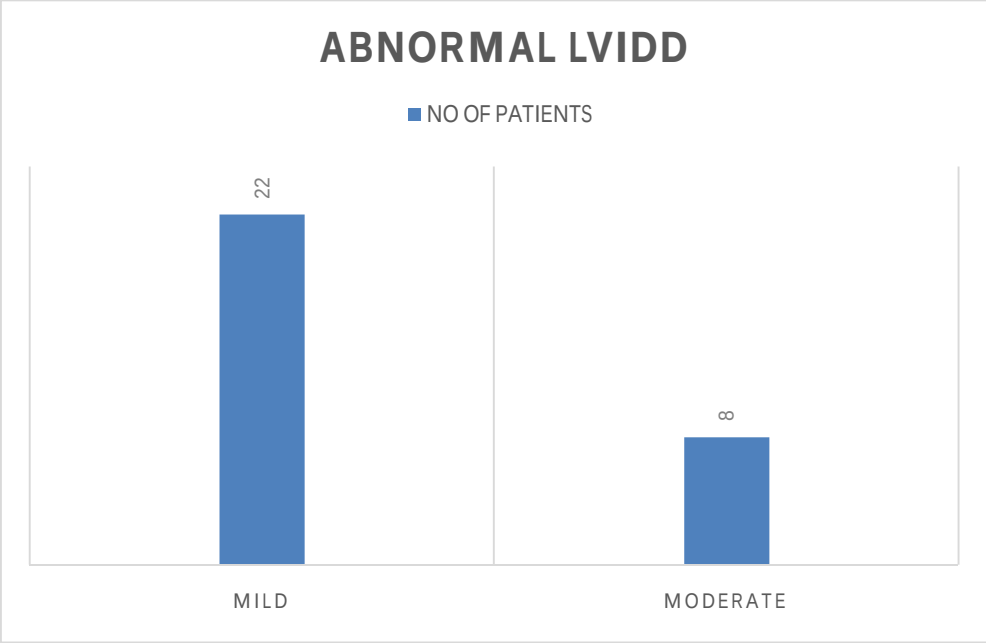


Table 20: Ejection fraction

EJECTION FRACTION	NO OF PATIENTS	PERCENTAGE
NORMAL	71	70%
LOW	29	30%

Chart 20: Ejection fraction

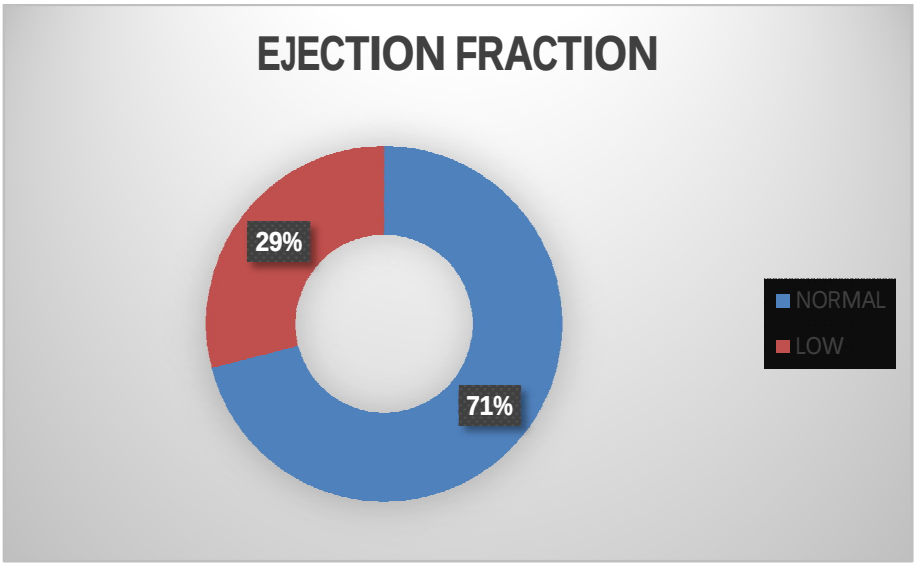
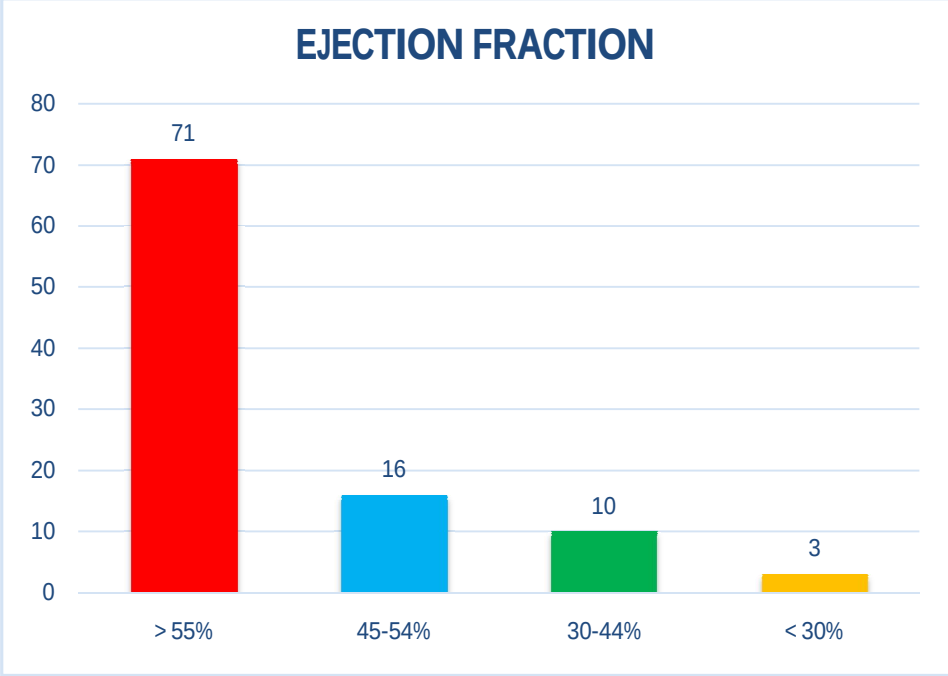


Table 21: Ejection Fraction

EJECTION FRACTION	NO OF PATIENTS	PERCENTAGE
> 55%	71	71%
45-54%	16	16%
30-44%	10	10%
< 30%	3	3%

Chart 21: Ejection Fraction

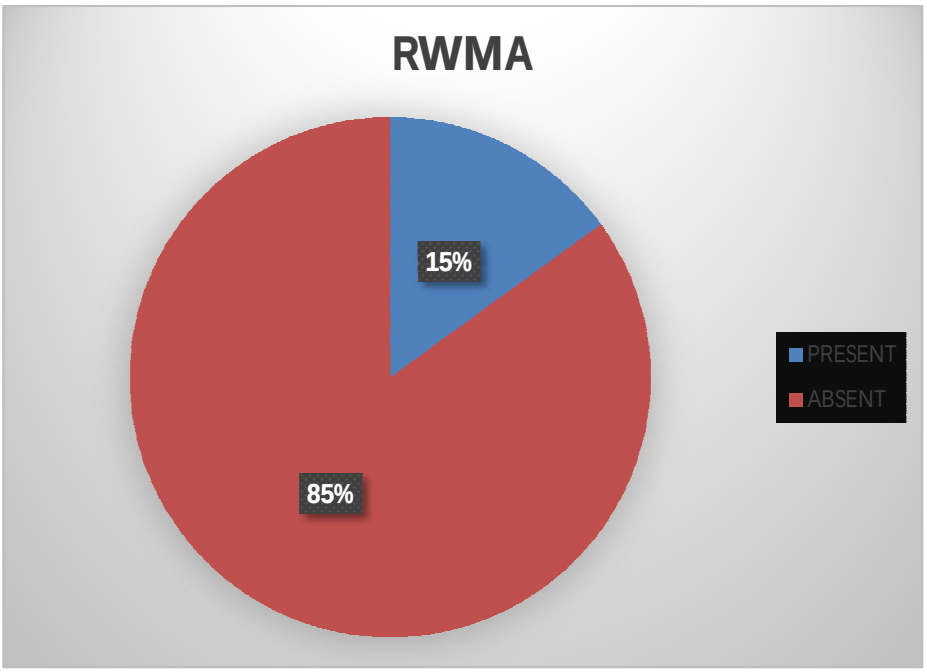


In our study group decreased ejection fraction was seen in 29 patients amog which 16 aptients had mild decrease, 10 patients had moderate decrease and 3 had severe reduction

Table 22: Regional wall motional abnormality

RWMA	NO OF PATIENTS	PERCENTAGE
PRESENT	15	15%
ABSENT	85	85%

Chart 22: Regional wall motional abnormality

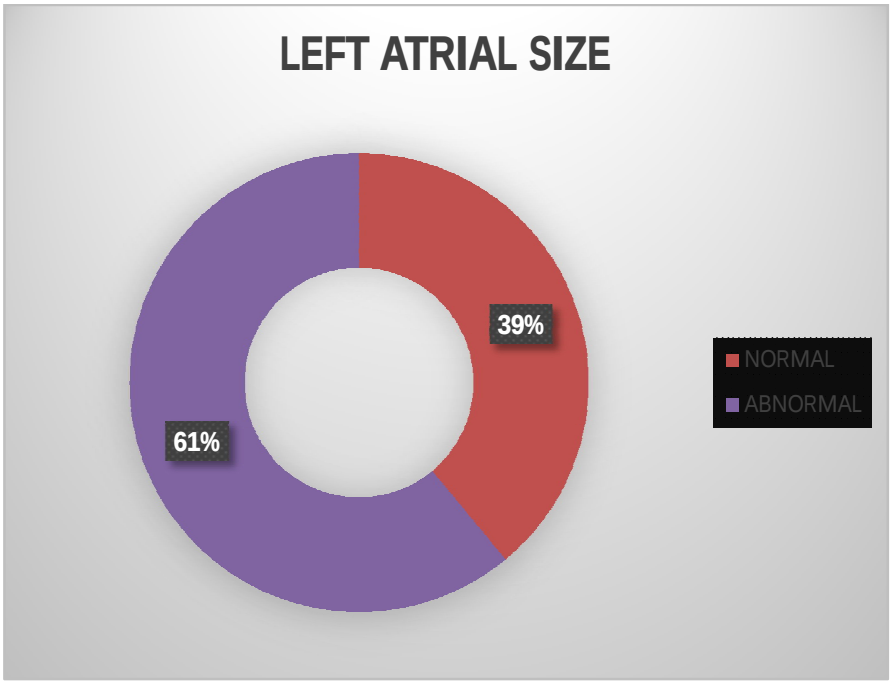


Regional wall motion abnormality was seen in 15 patients in our study group.

Table 23: Left atrial size

LEFT ATRIAL SIZE	NO OF PATIENTS	PERCENTAGE
NORMAL	39	39%
ABNORMAL	61	61%

Chart 23: Left atrial size



Left atrial size is increased in 61 patients among which it was mildly increased in 41 patients, moderately increased in 14 patients and severely increases in size in one patient.

Table 24: Left atrial size

LA SIZE	NO OF PATIENTS	PERCENTAGE
NORMAL	39	39%
MILDLY ABNORMAL	46	46%
MODERATELY ABNORMAL	14	14%
SEVERELY ABNORMAL	1	1%

Chart 24: Left atrial size

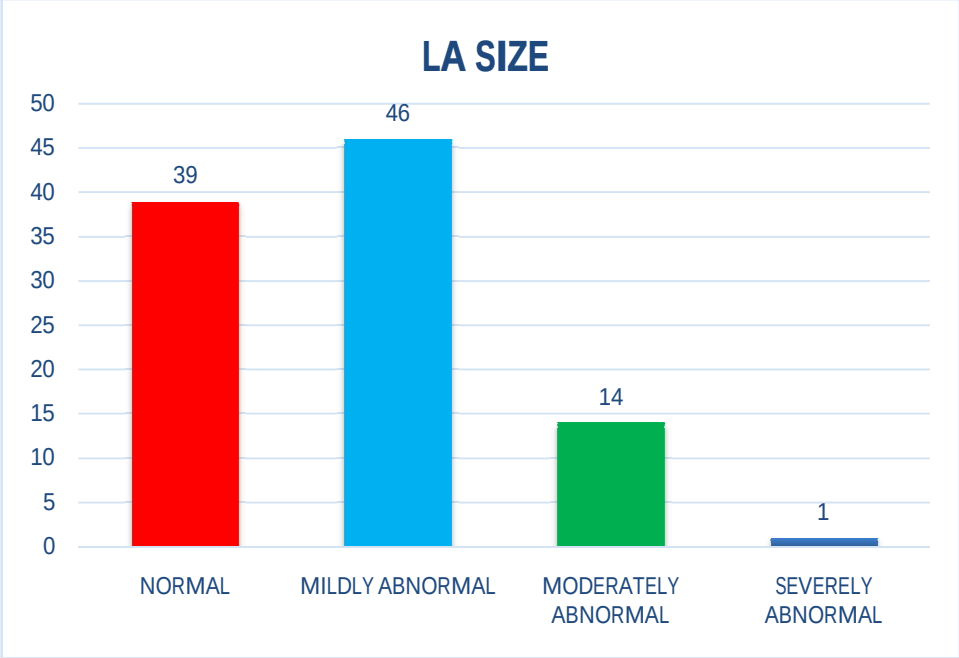
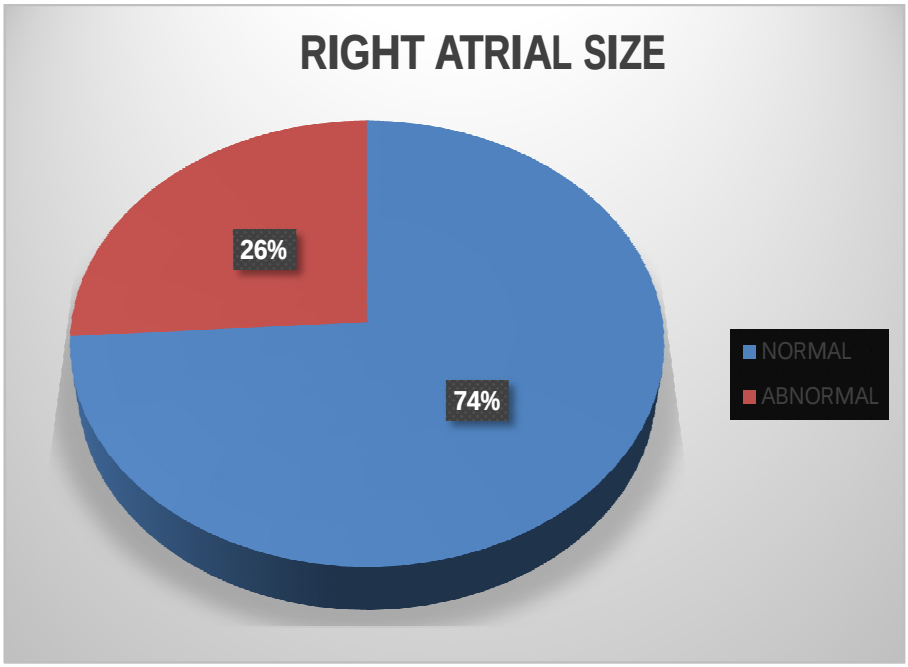


Table 25: Right atrial size

RIGHT ATRIAL SIZE	NO OF PATIENTS	PERCENTAGE
NORMAL	74	74%
ABNORMAL	26	26%

Chart 25: Right atrial size

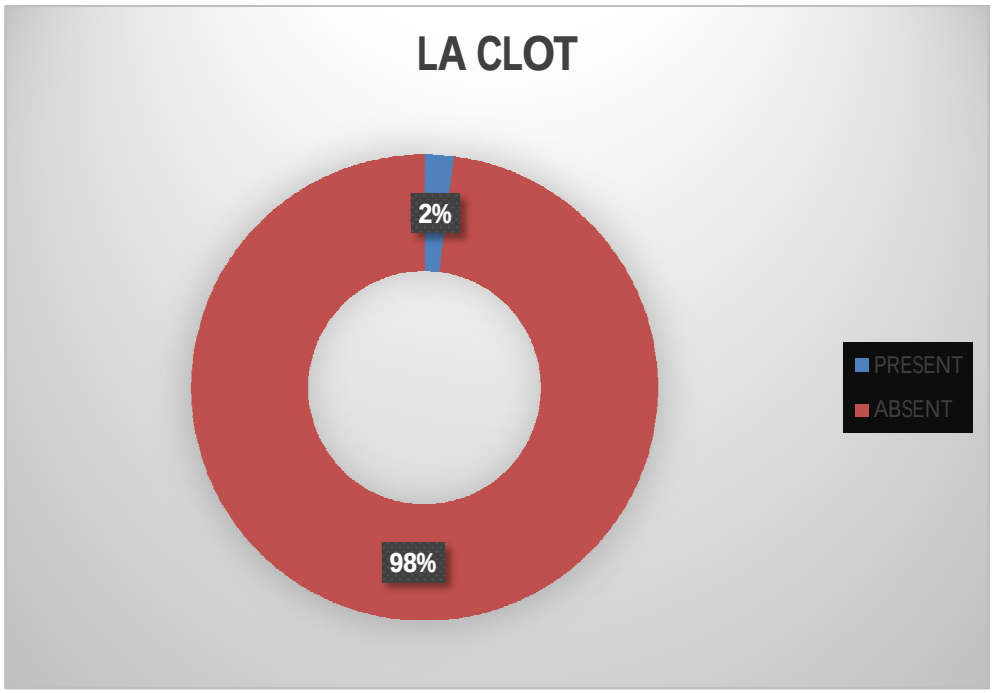


Right atrial size was increased in 26 percent of patients in our study group.

Table 26: Left atrial clot

LA CLOT	NO OF PATIENTS	PERCENTAGE
PRESENT	2	2%
ABSENT	98	98%

Chart 26: Left atrial clot



Left atrial clot was seen in only 2 patients in our study group.

DISCUSSION

In our study 100 patients who satisfied the inclusion and exclusion criteria with Atrial Fibrillation were included, the mean age of our study population was 47.64 years. In our study we divided the patients into 4 Age groups, as follows < 20 years, 2. 21-40 years, 41-60 years – 30,> 60 years. In our study maximum number of patients were in the 41 -60 years age group followed by 21- 40 years age group while it was much less in other age group.

This distribution is in contrast with previous studies done where the prevalence of AF shows a strong age dependence varying from 0.5% in patients aged <40 years to 5% in patients aged >65 years and nearly 10% amongst octogenarians [3–6]. Both the Framingham Heart Study and the Rotterdam Study estimated that the lifetime risk for development of AF in adults >40 years and at the age of 55 years respectively to be approximately 1 in 4.Novel risk factors for AF in young age are increasingly being discovered such as genetic causes, lifestyle factors (such as alcohol consumption, personality traits, and smoking), body mass index, and physical activity. In addition, it can also be argued that occult cardiac pathologies such as hypertension or ischemic heart disease may well be diagnosed in these patients if they are investigated thoroughly. A decreased mean age in our study may be due to above associated factors among patients in our study.

In our study females were more in number than males. Out of 100 total patients 67 were females 33 were males. The male: female ratio was 2:1 in contrary to normal pattern. In general male patients are moreaffected than

female patients in AF but in our study female patients are more affected, since most common disease associated with our study population is rheumatic heart disease there may female preponderance ,this results was similar to study done by Dushyant S., et al52.

Among our patients 63 percent had past history of RHD and on treatment for the same. In our Study Rheumatic heart disease was the most common etiologicalfactor associated with AF. These results are similar to previous studies done by Kannel WB., et al and Diker E., et al 48,50. One-fourth of patients with rheumatic valvular heart disease have Atrial Fibrillation (AF) [5]. Probability of development of AF is high in patients with MS, owing to Left Atrial (LA) dilatation in response to valve obstruction and the inflammatory and fibrotic changes caused by the rheumatic process [6,7]. With the onset of AF, there is an unexpected loss of the atrial involvement to ventricular filling and about 30% reduction in cardiac output

The second most common predisposing factor in our study was systemic hypertension. Around 29 patients had history of treatment for SHT. This is similar with the study by Framingham76, who also found a significant association between SHT and AF. HTN is the most common cardiovascular disorder and AF is the most common clinically significant arrhythmia. Both conditions are associated with aging and often coexist.17–20 In some studies, up to 90% of AF patients are observed to be hypertensive (Figure 1).21–23 Beyond the direct relations between AF and HTN, HTN is also associated with other cardiovascular co morbidities that increase risk for AF, including

coronary artery disease, heart failure, metabolic syndrome, chronic kidney disease, and sleep apnea.24–27Higher pulse pressure has also been shown to increase the risk of developing AF.28 In a prospective study involving Framingham Heart Study and Offspring participants, each 20 mm Hg increase in pulse pressure was associated with a 24% increased risk of AF over a 20-year follow-up. Notably, models were adjusted for mean blood pressure and other clinical risk factors for both AF and HTN. This suggests that increased pulse pressure may be an independent predictor of arterial stiffness and capture an additional modifiable AF risk element distinct from systolic hypertension.28 During our evaluation 32 percent of patients had an elevated blood pressure while the rest of them had normal or low blood pressure.

Also about 17 patients with AF in our study had CAD as a possible etiological factor, and this conforms to the study by Kannel WB., et al and Crenshaw BS., et al48,49. The prevalence of CAD in patients with AF is from 17% to 46.5% which is similar to our study while the prevalence of AF among patients with CAD is low and it is estimated from 0.2% to 5%.

In our study group around 22 patients had history of treatment for diabetes mellitus and 13 patients had past history of COPD. COPD is not only an independent predictor for major adverse cardiac events but also a predictor of AF incidence [3, 4]. The rate of AF incident was inversely associated with forced expiratory volume in one second (FEV1). AF prevalence was higher in severe airflow obstruction subject than those with mild or moderate airflow obstruction [5, 6]. Moreover, the risk of AF hospitalization was higher among

the lower FEV1, especially for FEV1 < 60% [7]. Previous Studies have reported that the major cause of deaths in COPD patients is cardiovascular diseases (CVD) rather than respiratory failure and it is more likely to develop acute coronary syndrome and heart failure. Furthermore, COPD contributes most to the increased all-cause mortality of AF [8].

Among other diseases associated in patients with atrial fibrillation.In our study group thyroid disorders were seen in 5 patients(hyperthyroidism -2 & Thyrotoxicosis -3). AF occurs in up to 15% of patients with hyperthyroidism compared to 4% of people in the general population and is more common in men and in patients with triiodothyronine (T3) toxicosis. The incidence of AF increases with advancing age. Also, subclinical hyperthyroidism is a risk factor associated with a 3-fold increase in development of AF. Thyrotoxicosis exerts marked influences on electrical impulse generation (chronotropic effect) and conduction (dromotropic effect). Several potential mechanisms could be invoked for the effect of thyroid hormones on AF risk, including elevation of left atrial pressure secondary to increased left ventricular mass and impaired ventricular relaxation, ischemia resulting from increased resting heart rate, and increased atrial ectopic activity. Re-entry has been postulated as one of the main mechanisms leading to AF. AF is more likely if effective refractory periods are short and conduction is slow. Hyperthyroidism is associated with shortening of action potential duration which may also contribute to AF

Different types of cardiomyopathy was seen in around 5 patients(HOCM-3,DCM-1 & RCM 1), Increased left atrial size and volume along with impaired

left atrial function confer an increased likelihood of AF. Atrial septal defect was seen in a solitary patient.

Coming to presenting complaints an previous study done by Flaker, Greg C., et al study70 showed 78% of the patients with breathlessness and next commonest presentation was chest pain. Similarly another study done by Tischler et al 75 showed breathlessness in 62%of patients,' palpitation' in 33% patients, and giddiness in 12% patients, In our study predominant symptom was breathlessness similar to above studies with 78 patients presented with breathlessness followed by palpitation in 53% cases, rest of the common symptoms were chest pain (n=26) and pedal edema (n=35). Giddiness was seen only in six patients, weakness of limb in seven patients.

Most of the patients actually had multiple symptoms.In our study 32 patients presented with single complaints, while patients who had two complaints were also 32. Rest of 36 patients presented with two or more complaints.

In our study personal habits like smoking and alcohol intake was seen in around 25% of cases. Smoking habit was seen in 16 patients and alcohol intake in 19 patients. Cigarette smoking and excessive drinking are important risk factors for incidence of AF

Cardiac failure is seen in 37 percent of patients in our study group.We also evaluated the ejection fraction of all patients in our study group, In our study 71 % of the patients had normal ejection fraction and 29 patients had reduced EF Among that 16% of the patients had mildly abnormal,10% of the

patients had moderately abnormal, and the remaining 3% of them presented with severely abnormal EF, Presence of such abnormal EF(LV systolic dysfunction) independently predicts the risk of stroke shown by study on atrial fibrillation by researchers 69. The reported prevalence of AF in heart failure series ranges from 13% to 27%.12–16 In the Framingham Heart Study, 26% of patients developed both AF and heart failure.17 Moreover, the prevalence of AF in patients with heart failure increased in parallel with the severity of the disease, ranging from 5% in patients with mild to 10% to 26% among patients with moderate up to 50% in patients with severe heart failure.18 This results are in similar to our study.

Next we evaluated the heart rate in our patient with 46% of patients had normal heart rate were as 5 patients had bradycardia and around 49% of patients had tachycardia. As we all know AF is conditions with irregularly irregular rate, it may be fast sometimes nu not necessarily.

Further we evaluated the transthoracic echocardiographic finding in all patients of our study group

To start with first let’s discuss the Left ventricular internal dimension during diastole which was abnormal i.e. Increased in 30 percent of patients in our study group. Among which 22 patients had mild increase in diameter and 8 patients had moderate increase in diameter. 4 Increased LV size as detected by echocardiography is a strong independent predictor of cardiovascular morbidity, especially in hypertensive men. In previous studies age and body weight and cardiovascular features such as the duration of atrial fibrillation, LV mass,

annular calcification, the severity of coronary artery disease, and hypertension have been related to left ventricular diameter [6-10].

Next we evaluated the regional wall motion abnormality which was seen in 15 patients in our study group. Regional wall motional abnormality has a direct influence on ejection fraction and left atrial size which in turn becomes a risk factor for developing AF.

The left atrial size was abnormal in most of patients in our study group with around 61% patients having an abnormal sized left atrium. Among these 61 patients there was mild increase in 46 patients, moderate increase in 14 patients and severe increase in one patient. Occurrence of AF is known to correlate with LA size; the incidence of AF rises from 3% when the left atrial diameter is < 40mm to 54% if the left atrial diameter is > 40 mm. The left atrium (LA) has a reservoir and conduit function, and is an important regulator of left ventricular filling.[3] It also reflects an important electrophysiological substrate and has neurohumoral properties by releasing natriuretic peptides.[3,5] Those factors are closely related to left ventricular systolic and diastolic function.[3,6] LA enlargement itself is an important risk factor for incident AF[6–10], and is related to an increased stroke risk.[6–12] LA size is also a key determinant for the success of rhythm control strategies in patients with AF.[3,4]

Similarly we evaluated the right atrial size which was increased only in 26 patients in our study group. Atrial structural or electrical remodelling is commonly observed in patients with AF. However, this knowledge is mainly

based on LA studies, whereas little is known about the potential relationship between RA disease and pathophysiology of AF. Anatomical and histological changes in the RA in patients with AF have been recognized with recent advances in cardiac imaging tools and electrophysiology and a study done reported that about 1 in 5 AF events have a RA origin. Hence in our study too there is not much patients who had right atrial enlargement, which is comparatively of less incidence than left atrial enlargement.

Left atrial clot was seen in only 2% of patients in our study group. This is similar to previous studies done where the incidence was around 1.5% among AF patients

SUMMARY

- Atrial Fibrillation is the one of the most commonly encountered weak or irregular heart beat (arrhythmia) in our population.
- This study was undertaken to study about clinical profile of Atrial fibrillation and its transthoracic echocardiography presentation.
- A total of 100 patients who satisfied inclusion and exclusion criteria with atrial fibrillation were taken up for study,
- After ethical committee clearance, and obtaining an informed consent, the patient's History, clinical and laboratory data were collected and analysed statistically.
- In our study females were more in number than males. Out of 100 total patients 63 were females, and 37 were males with a male: female ratio of 1:1.7.
- Most common age group was 41-60 years.
- Rheumatic heart disease was the most common cause of AF in our study,
- Systemic hypertension was the second most common cause for AF.
- CAD was seen in 17 cases, Diabetes in 22 cases and COPD in 13 cases
- Breathlessness was the most common complaint followed by palpitation.
- Smoking habit seen in 16 patients and alcohol intake in 19 patients.
- Cardiac failure seen in 37 percent of cases while ejection fraction was decreased in 29 patients.
- Uncontrolled heart rate or tachycardia is seen in 49 percent of patients.

- Coming to ECHO findings increased LVIDd was seen in 30 patients
- Regional wall motion abnormality seen in 15 percent of patients in our study group.
- Increased left atrial size – the most common ECHO finding seen in atrial fibrillation was seen in 61 patients while increased right atrial size was seen in 26 patients.
- We detected LA Clot only in a very few patients withAF because TTE has low sensitivity of detecting LA clot.

CONCLUSION

To conclude from above study its clear rheumatic heart disease was found to be the most common cause of atrial fibrillation followed by dilated cardiomyopathy. Females were more affected as compared to males.Common presentations were shortness of breath, palpitations.Commonest finding in ECHO is increased left atrial size. A complete echocardiographic evaluation is must in patient diagnosed with atrial fibrillation, which helps in deciding the prognosis of the patient also helps in assessing the chances of developing thrombo-embolic episodes. Hence regular monitoring with ECHO is advisable in AF patients.

ANNEXURE

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Proforma

Clinico-etiological profile of Atrial Fibrillation and its correlation with Atrial size

Name
Age
Sex
Address

Presenting complaints
1.Palpitation
2.Giddiness/ Syncope
3.Chest pain
4. Breathlessness
5.Pedaledema
6.Weakness of limbs
7. others

Past history:
1.RHD
2.CAD
3.SHTN
4.DM
5.COPD
6.Others

Personal history:
1.Alcoholic
2.Smoker
3.Substance abuse

General examination:
Consciousness:
Pallor :
Clubbing:
Pedal edema:
Elevated JVP:
Signs of Hyperthyroidism:
Vitals:
Pulse Rate:
Pulse Deficit:
Blood Pressure

Systemic Examination:
CVS:
RS:
P/A:
CNS:

Investigation:
Hb:
SERUM ELECTROLYTES:
Lipid profile:
TFT (optional):
ECG:
Chest X-ray:
Echocardiogram (Transthoracic)
LVIDd
LVIDs
EF:
RWMA:
MITRAL VALVE: MS: MR
AORTIC VALVE:
LEFT ATRIAL SIZE
RIGHT ATRIAL SIZE:
LA CLOT/ VEGETATION:

DIAGNOSIS:

MASTER CHART

S.NO	AGE	AGE GROUP	SEX	SYMPTOMS	RHD	CAD	SHTN	DM	COPD	OTHERS	HABITS	FAILURE	HEART RATE	BP	LVI Dd	EF	RWMA	LA SIZE	RA SIZE	LA CLOT
1	59	3	M	1+2	N	N	Y	N	Y	N	2	N	2	1	2	1	N	1	2	N
2	38	2	F	1+3+5	Y	N	N	N	N	N	4	Y	3	2	1	1	N	3	2	N
3	45	3	F	1+5	Y	N	N	Y	N	N	4	N	2	2	1	2	N	2	1	N
4	65	4	M	1+3	N	Y	Y	Y	Y	N	1+2	Y	2	1	3	3	Y	2	2	N
5	28	2	F	4	Y	N	N	N	N	N	4	N	3	2	1	1	N	2	1	N
6	40	2	M	1+4+5	Y	N	N	N	N	N	1	Y	3	2	1	1	N	2	1	N
7	20	1	F	4	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
8	58	3	F	1+2+3+6	Y	Y	Y	Y	N	N	4	Y	3	1	2	3	Y	3	2	N
9	58	3	M	1+3+4	Y	N	N	Y	N	N	4	Y	3	2	1	1	N	2	1	Y
10	35	2	F	3+4	Y	N	N	N	N	N	4	N	2	2	1	1	N	2	1	N
11	70	4	M	2+4+5	N	N	Y	Y	Y	N	1+2	N	1	1	2	2	N	2	1	N
12	55	3	F	2+4+6	N	Y	Y	Y	N	N	4	Y	2	1	3	3	Y	3	2	N
13	45	3	M	1+4+5	Y	N	N	N	N	N	4	Y	3	2	1	1	N	3	2	N
14	33	2	F	1+4	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
15	38	2	F	3+4+5	Y	N	N	N	N	N	4	Y	3	2	1	1	N	2	1	N
16	84	4	M	1+4+6	N	Y	Y	N	Y	N	1+2	Y	3	1	2	2	Y	2	2	N
17	52	3	F	4	Y	N	Y	N	N	N	4	N	2	1	1	1	N	1	1	N
18	32	2	F	1+3+4	Y	N	N	N	N	N	4	N	2	2	1	1	N	2	1	N
19	45	3	F	1+4	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
20	59	3	F	1+4	Y	N	Y	N	N	N	4	Y	3	1	1	1	N	2	1	N
21	29	2	F	4	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N

22	67	4	M	3+4+5	N	N	Y	N	Y	N	2	N	3	1	2	2	N	2	1	N
23	39	2	M	1+4+5	Y	Y	Y	N	N	N	4	Y	3	2	1	2	Y	1	1	N
24	54	3	F	3+4+5	N	Y	Y	Y	N	N	4	Y	3	2	2	3	Y	3	2	N
25	65	4	M	6	N	N	Y	N	N	N	1+2	N	2	1	2	2	N	2	2	N
26	45	3	F	1	N	N	N	N	N	HYPERTHYROID	4	N	2	2	2	1	N	1	1	N
27	40	2	F	4+5+7	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
28	58	3	M	1+4	N	N	N	N	N	N	1	N	2	2	1	1	N	2	1	N
29	39	2	F	4	Y	N	N	N	N	N	4	N	3	2	1	1	N	2	1	N
30	33	2	M	4	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
31	48	3	M	1+4+5	Y	N	N	N	N	N	2	N	2	2	1	1	N	1	1	N
32	42	3	M	1+3+4+5	Y	N	N	N	N	ASD	4	Y	2	2	1	2	N	2	2	N
33	50	3	M	1+4	N	Y	N	N	N	DCM	4	N	1	2	1	3	Y	3	2	N
34	35	2	M	4	N	Y	N	N	N	N	4	N	1	2	2	2	N	2	1	N
35	37	2	F	1+4+5	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
36	59	3	F	6	Y	Y	N	Y	N	N	4	N	3	1	2	2	N	2	1	N
37	56	3	F	5	N	N	Y	Y	Y	N	4	Y	3	2	2	2	N	1	2	N
38	70	4	F	1+5	N	Y	N	N	N	N	4	N	3	2	3	3	Y	2	2	N
39	80	4	M	1+4+5	N	Y	Y	Y	Y	N	1+2	N	2	1	2	3	Y	2	2	N
40	28	2	F	4	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
41	45	3	F	4+5	N	N	N	N	Y	N	4	Y	2	2	2	2	N	2	2	N
42	61	4	F	1+5	N	N	N	N	N	HYPERTHYROIDISM	4	N	3	2	1	1	N	1	1	N
43	21	1	F	4	Y	N	N	N	N	N	4	N	2	2	1	1	N	2	1	N
44	52	3	M	3+4+5	N	N	N	N	N	RESTRICTIVE CARDIOMY	2	Y	1	2	1	3	Y	3	2	N

										POPATHY										
45	46	3	F	4+5	Y	N	N	Y	N	N	4	Y	2	2	1	1	N	1	1	N
46	28	2	F	4	Y	N	N	N	N	N	4	N	3	2	1	1	N	1	1	N
47	32	2	F	1+4	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
48	52	3	M	1+4+5	Y	N	N	Y	N	N	2	Y	3	3	2	2	N	2	1	N
49	26	2	F	4	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
50	40	2	M	1+2	N	N	N	N	N	N	1+2	N	2	2	1	1	N	1	1	N
51	40	2	M	3+4	Y	N	N	N	N	N	1	N	2	2	1	1	N	1	1	N
52	45	3	F	1+4	Y	N	N	Y	N	N	4	N	2	2	1	1	N	1	1	N
53	52	3	F	1+4+5	Y	N	N	N	N	N	4	Y	2	1	1	1	N	2	1	N
54	32	2	F	1+4	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
55	58	3	F	1+4+5	Y	N	Y	Y	N	N	4	Y	2	2	1	1	N	2	1	N
56	40	2	F	4	Y	N	N	N	N	N	4	N	3	2	1	1	N	1	1	N
57	56	3	F	1+4	Y	N	N	N	N	N	4	N	3	1	1	1	N	2	1	N
58	58	3	M	1+5	N	N	Y	N	N	N	1	N	3	2	1	1	N	1	1	N
59	70	4	M	7	N	N	N	N	N	N	4	N	3	2	1	1	N	2	1	N
60	54	3	F	1+3+4+5	N	Y	N	N	N	DCM	4	Y	3	2	3	4	Y	3	2	N
61	40	2	M	4	Y	N	N	N	N	N	1	N	2	2	1	1	N	1	1	N
62	32	2	F	4	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
63	55	3	M	1+3+4+5	N	Y	Y	N	N	N	4	Y	3	1	2	1	N	2	1	N
64	60	3	F	1+4	N	N	Y	N	Y	N	4	Y	2	1	2	1	N	1	1	N
65	49	3	F	4	Y	N	N	N	N	N	4	N	1	1	1	1	N	1	1	N
66	55	3	M	1+3+4	N	N	N	N	N	THYROTOXICOSIS	2	Y	3	2	1	2	N	2	2	N
67	39	2	F	1+4	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N

68	49	3	M	7	N	N	N	N	N	N	1+2	N	2	2	1	1	N	1	1	N
69	69	4	M	3+4+5	N	N	N	N	Y	N	2	Y	3	2	1	1	N	1	2	N
70	30	2	F	4	Y	N	N	N	N	N	4	N	2	3	1	1	N	1	1	N
71	45	3	F	3+4+5	Y	N	Y	Y	N	N	4	Y	3	1	2	2	N	2	1	N
72	60	3	F	1+4+5	N	Y	Y	Y	N	DCM	4	Y	3	1	3	3	Y	3	2	N
73	30	2	M	1+4	N	N	N	N	N	ASD	4	N	3	2	1	1	N	3	2	N
74	39	2	F	3+4+5	Y	N	N	N	N	N	4	Y	3	1	1	1	N	2	1	N
75	55	3	F	3+4	N	Y	Y	Y	Y	N	4	Y	3	2	3	4	Y	3	2	N
76	32	2	F	4	Y	N	N	N	N	N	4	N	2	1	1	1	N	2	1	N
77	40	2	F	1+3+4	Y	N	N	N	N	N	4	N	3	2	1	1	N	2	1	N
78	50	3	F	4	Y	N	Y	N	N	N	4	N	3	2	1	1	N	2	1	N
79	36	2	F	1	Y	N	N	N	N	N	4	N	2	2	1	1	N	1	1	N
80	65	4	M	1	N	N	Y	N	N	N	2	N	3	1	2	1	N	2	1	N
81	49	3	F	1+3+5	Y	N	N	N	N	N	4	Y	3	1	1	1	N	2	1	N
82	70	4	M	4	N	N	N	N	Y	N	2	Y	3	1	2	1	N	2	2	N
83	37	2	F	4	Y	N	N	N	N	N	4	N	3	2	1	1	N	2	1	N
84	60	3	F	1+4	N	N	N	N	Y	N	4	N	3	1	2	1	N	2	2	N
85	45	3	M	1+3+4+5	Y	N	Y	N	N	N	4	Y	3	1	1	1	N	3	1	N
86	55	3	M	1	N	N	N	N	N	N	1+2	N	3	1	2	1	N	2	1	N
87	70	4	F	1+6+7	Y	N	Y	N	N	N	4	N	3	1	1	2	N	2	1	N
88	30	2	F	4	Y	N	N	N	N	N	4	N	2	1	1	1	N	1	1	N
89	50	3	M	1+4+5	Y	Y	Y	Y	N	N	1	Y	3	2	3	3	Y	3	2	N
90	44	3	M	3+4	N	Y	Y	Y	N	N	1+2	Y	3	2	3	4	Y	4	2	Y
91	3	2	F	4+5	Y	N	N	N	N	N	4	Y	3	2	1	1	N	1	1	N

	9																			
92	50	3	F	1+4	Y	N	N	Y	N	N	4	N	2	2	1	1	N	1	1	N
93	35	2	F	3+4	Y	N	N	N	N	N	4	N	3	2	1	1	N	2	1	N
94	47	3	F	3+4	Y	N	N	Y	N	N	4	N	2	2	1	1	N	1	1	N
95	54	3	F	4	Y	N	N	N	N	N	4	N	2	2	1	1	N	2	1	N
96	70	4	M	1+2	N	N	Y	Y	N	N	1+2	N	3	2	1	1	N	2	1	N
97	33	2	F	1+4+5	Y	N	N	N	N	N	4	Y	3	1	1	1	N	3	1	N
98	54	3	M	1+3+4	Y	N	N	N	N	N	4	Y	3	2	1	1	N	2	1	N
99	62	4	F	1+6	N	N	Y	N	N	N	4	N	2	1	2	2	N	2	1	N
100	40	2	F	4	Y	N	N	N	N	N	4	N	2	1	1	1	N	1	1	N

KEY TO MASTER CHART:

AGE:
<20 - 1
20-39 - 2
40-59 - 3
>60- 4

Presenting complaints:
1.Palpitation
2.Giddiness/ syncope
3.Chest Pain
4.Breathlessness
5.Pedal edema
6.Weakness of limbs
7. others

Past history:
YES- Y; NO-N
1.RHD
2.CAD
3.SHTN
4.DM
5.COPD
6.Others

Personal history:
1.Alcoholic
2.Smoker
3.Substance abuse
4.No

Cardiac failure : yes-Y ; no-N
Heart rate:
1.Slow: (<80/MIN)
2.Controlled (80-110/MIN)
3.Uncontrolled (rapid) (110/MIN)

Blood pressure
1.Hypertension (>140/90 mm of Hg)

2.normal

Echo :

LVIDd(Left ventricular internal Diameter in cm during diastole):-

MEN	**WOMEN**
Reference range: 4.2-5.9 → 1	3.9-5.3→1
Mildly abnormal: 6.0-6.3 → 2	5.4-5.7→2
Moderately abnormal: 6.4-6.8→3	5.8-6.1→3
Severely abnormal: ≥ 6.9→4	≥ 6.2→4

Ejection fraction:
Reference range:
≥ 55→1
45-54→2
30-44→3
< 30→4

RWMA:
Yes – Y
No- N

LA SIZE(Left Atrium Diameter in cm)

MEN	WOMEN
Reference range: 3.0-4.0→ 1	2.7-3.8→1
Mildly abnormal: 4.1-4.6→ 2	3.9-4.2→2
Moderately abnormal: 4.7-5.2→3	4.3-4.6→3
Severely abnormal : ≥ 5.3→ 4	≥4.7 →4

RA SIZE(Right Atrium Major Dimension in cm):-
Normal < 5.3 → 1
Abnomal > 5.3 → 2

LA Clot :-
YES- Y
NO- N

ETHICAL COMMITTEE APPROVAL

Ref.No.50/ME2/2017

Office of the Dean
Kanyakumari Govt. Medical College
Asaripallam 629 201

Dated: 20.12.2017

CERTIFICATE OF ETHICAL COMMITTEE APPROVAL

The Institutional Ethical Committee meeting was conducted on 20.12.2017 at 10.00 am at Medical Education Unit, Kanyakumari Govt. Medical College Asaripallam, to give approval of your study title "Clinico – etiological profile of Atrial Fibrillation in a tertiary hospital and its correlation to Atrial size"

The following members of the Ethical Committee attended the Meeting.

1.	Chairperson	: Dr. M. Kannan. M.D., Prof of Anaesthesia (Retired)
2.	Member Secretary	: Dr. T. Ashok Kumar M.D., Prof & HOD of Pharmacology , Kanyakumari Govt. Medical College Asaripallam
3.	Basic Medical Scientists	: 1. Dr. R. Rajesh M.D., Assoc. Professor & HOD of Forensic Medicine Kanyakumari Govt. Medical College Asaripallam 2. Dr. K.U. Suresh Balan MD Associ. Prof & HOD of Community Medicine Kanyakumari Govt. Medical College Asaripallam
4.	Clinicians	:1. Dr. Usha M.S Prof. & HOD of Surgery Kanyakumari Govt. Medical College Asaripallam 2. Dr. Prince Sree Kumar Pius MD Prof. of Medicine Kanyakumari Govt. Medical College Asaripallam 3. Dr. A.J.S Pravin M.D Prof. & HOD of Dermatology Kanyakumari Govt. Medical College Asaripallam 4. Dr. J.A. Jayalal M.S Assoc. Prof. of Surgery, Kanyakumari Govt. Medical College Asaripallam 5. Dr. Edward Johnson MD Assoc. Prof. of Anaesthesia, Kanyakumari Govt. Medical College Asaripallam

5.	Representative of Non Government Voluntary Agency	:	Mrs. Jesintha Dharma
6.	Theologian	:	Dr. Surendra M.A, M.Phil, M.Ed.,Ph.D.,
7.	Lay Person	:	Thiru. Justin

The committee has given approval of your study subject to the following conditions

- The Study should be conducted in its presented form.
- The Progress of the study and any changes in the study to be informed to the committee
- Copy of the final result of study may be furnished to the committee.

Member Secretary
Institutional Ethical Committee
Kanyakumari Govt Medical College
Asaripallam

MEMBER SECRETARY
INSTITUTIONAL ETHICAL COMMITTEE
KANYAKUMARI GOVT MEDICAL COLLEGE
ASARIPALLAM-629 201

Chairman
Institutional Ethical Committee
Kanyakumari Govt Medical College
Asaripallam

CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
KANYAKUMARI GOVT. MEDICAL COLLEGE
ASARIPALLAM-629 201

To

Dr. Suhas Raj S
PG in MD General Medicine
Dept. of Gen. Medicine
KGMC Asaripallam

CONSENT

கன்னியாகுமரி அரசு மருத்துவக்கல்லூரி மருத்துவமனை ஆசாரிப்பள்ளம்
நாகர்கோவில்

பொது மருத்துவத்துறை

ஆராய்ச்சியில் பங்கு பெற ஒப்புதல் அளிக்கும் படிவம்

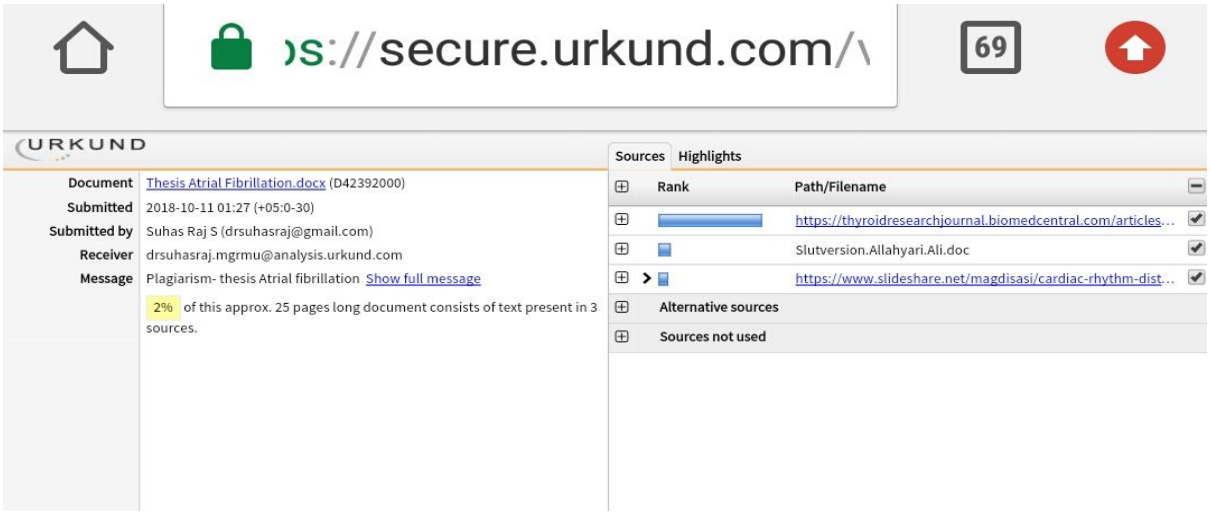
நான் ஏட்ரியல் குறுநடுக்கம் எனப்படும் (Atrial Fibrillation) ஒரு வித நெஞ்சுதுடிப்பு நோயால் பாதிக்கப்பட்டு இந்த ஆசாரிப்பள்ளம் மருத்துவக் கல்லூரி மருத்துவமனையில் சிகிச்சை பெற்று வருகிறேன். இதற்கான காரணத்தைக் கண்டறியும் ஆராய்ச்சியைப் பற்றி மருத்துவர் மூலம் அறிந்து கொண்டேன். இதற்காக மின் ஒலி இதய வரைவி (Echo) எனப்படும் பரிசோதனையை மேற்கொள்ள வேண்டும் என அறிந்தேன். மேற்கூறிய அனைத்தும் நன்கு புரிந்து கொண்டு இந்த ஆராய்ச்சியில் ஈடுபட முழு சம்மதம் அளிக்கிறேன்.

நோயாளியின் ஒப்பம் :

தேதி :

நோயாளியின் உறவினர் ஒப்பம் :

ANTI-PLAGIARISM



URKUND 0B/s

Urkund Analysis Result

Analysed Document: CLINICO-ETIOLOGICAL PROFILE OF ATRIAL FIBRILLATION IN A TERTIARY CARE HOSPITAL AND ITS CORRELATION TO ATRIAL SIZE.docx (D42565049)
Submitted: 10/15/2018 11:50:00 AM
Submitted By: drsuhasraj@gmail.com
Significance: 3 %

Sources included in the report:

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Slutversion.Allahyari.Ali.doc (D3554681)
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http://www.onlinejacc.org/content/64/21/2246
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https://www.bioportfolio.com/resources/pmarticle/1988288/Validation-of-Right-Atrial-Area-as-a-Measure-of-Right-Atrial-Size.html
https://thyroidresearchjournal.biomedcentral.com/articles/10.1186/1756-6614-2-4

Instances where selected sources appear:

14

ANTI-PLAGIARISM CERTIFICATE

CERTIFICATE - II

This is to certify that this dissertation work titled **CLINICO-ETIOLOGICAL PROFILE OF ATRIAL FIBRILLATION IN A TERTIARY CARE HOSPITAL AND ITS CORRELATION TO ATRIAL SIZE** of the candidate **Dr.S.Suhas Raj** with registration Number **111151** for the award of **M.D** in the branch of **GENERAL MEDICINE** . I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows ..2.... percentage of plagiarism in the dissertation.

15/10/2018

Guide & Supervisor sign with Seal.

Prof. Dr. Prince Sreekumar Pius, M.D.,
Reg. No. 47734
Professor of Medicine
KGMCH, Asaripallam